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# "THERAPEUTIC DENTISTRY"

***Educational and methodical textbook***
***for independent work of fifth-year students***
***of the dental faculty from therapeutic dentistry***

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The methodological reccommendations were prepared for the study of the program "Therapeutic dentistry" in the discipline "Therapeutic dentistry" for the 5th year students of the dental faculty. Methodological recommendations include methodological developments for conducting practical classes in conjunction with control tasks and a list of recommended educational and methodological literature. This methodological recommendations are designed to deepen students' knowledge of the diseases of oral mucosa aand ways of their treatment.

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Reviewer: MD, prof. Department of Therapeutic Dentistry Kuligina V.M.

**CONTENT**

# LIST OF ABBREVIATED TERMS

| | |
|---|---|
| OM | Oral mucose |
| MC | Mouth cavity |
| MM | Mucous membrane |

# INTRODUCTION

Modern methods of treatment of lesions of the oral mucosa and their complications require a dentist to have knowledge of the structure and function of the dentition, the latest materials, techniques and methods for their diagnosis and provision of quality dental care.

The material of this textbook will allow students to gain a thorough knowledge of the basic knowledge of the main diseases of the oral mucosa, instruments and their functions, methods of diagnosis and treatment of lesions of various nature, modern dental materials, and techniques for their use.

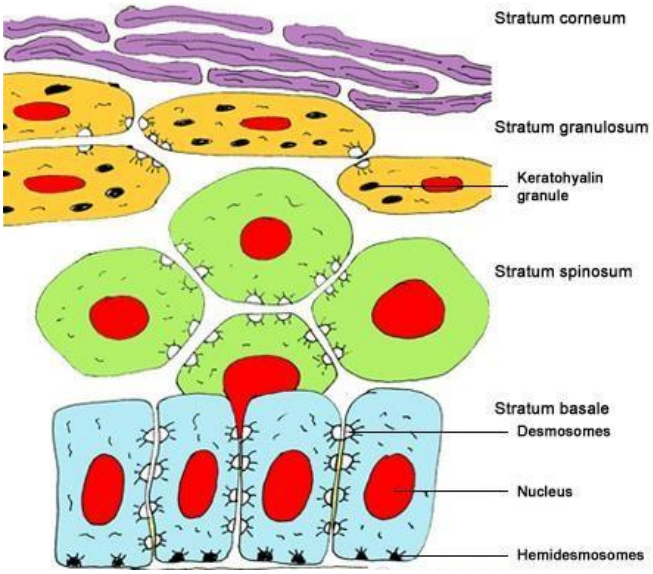
# TOPIC 1.

## ANATOMICAL AND HISTOLOGICAL FEATURES OF THE MUCOUS MEMBRANE OF THE ORAL CAVITY AND RED BORDER OF THE LIPS. PROTECTIVE FACTORS. SALIVA, ITS COMPOSITION AND PHYSIOLOGICAL ROLE.

The oral mucosa is the "skin" inside the mouth, and it covers most of the oral cavity apart from the teeth.

Function of oral mucosa

The oral mucosa has several functions. Its main purpose is to act as a barrier. It protects the deeper tissues such as fat, muscle, nerve and blood supplies from mechanical insults, such as trauma during chewing, and also prevents the entry of bacteria and some toxic substances into the body.




In general, maturation can undergo two different patterns:

1. Keratinisation; and

2. Non-keratinisation.The most common cells that are required to undergo cell turnover are called keratinocytes. As the keratinocyte matures, it undergoes modification in its structure that causes it to progress towards the surface of the epithelium, and eventually die. Keratinocytes mature to different degrees. In some areas of the mouth, the keratinocytes will fully mature (orthokeratinisation), whereas in other areas the keratinocytes will only partially undergo keratinisation (parakeratinisation).

Keratosis (from kerat- + -osis) is a growth of keratin on the skin or on mucous membranes stemming from keratinocytes, the prominent cell type in the epidermis.

Hyperkeratosis is thickening of the stratum corneum (the outermost layer of the epidermis, or skin), often associated with the presence of an abnormal quantity of keratin, and also usually accompanied by an increase in the granular layer. As the corneum layer normally varies greatly in thickness in different sites, some experience is needed to assess minor degrees of hyperkeratosis.

Parakeratosis is a mode of keratinization characterized by the retention of nuclei in the stratum corneum. In mucous membranes, parakeratosis is normal. In the skin, this process leads to the abnormal replacement of annular squames with nucleated cells. Parakeratosis is associated with the thinning or loss of the granular layer and is usually seen in diseases of increased cell turnover, whether inflammatory or neoplastic. Parakeratosis is seen in the plaques of psoriasis and in dandruff.

Granular parakeratosis (originally termed axillary granular parakeratosis) is an idiopathic, benign, nondisabling cutaneous disease that manifests with intertriginous erythematous, brown or red, scaly or keratotic papules and plaques. It presents in all age groups and has no established clinical associations.

Dyskeratosis is abnormal keratinization occurring prematurely within individual cells or groups of cells below the stratum granulosum.

Acantholysis is the loss of intercellular connections, such as desmosomes, resulting in loss of cohesion between keratinocytes, seen in diseases such as pemphigus vulgaris. It is absent in bullous pemphigoid, making it useful for differential diagnosis.

Acanthosis is diffuse epidermal hyperplasia (thickening of the skin, and not to be confused with acanthocytes). It implies increased thickness of the Malpighian layer (stratum basale and stratum spinosum)

Spongiosis is mainly intercellular edema (abnormal accumulation of fluid) in the epidermis, and is characteristic of eczematous dermatitis, manifested clinically by intraepidermal vesicles (fluid-containing spaces), "juicy" papules, and/or lichenification

Papillomatosis of skin is skin surface elevation caused by hyperplasia and enlargement of contiguous dermal papillae.

Primary lesions:

Macule: A macule is a change in surface color, without elevation or depression and, therefore, nonpalpable, well or ill-defined, variously sized, but generally considered less than either 5 or 10 mm in diameter at the widest point.

Patch: A patch is a large macule equal to or greater than either 5 or 10 mm across, depending on one's definition of a macule. Patches may have some subtle surface change, such as a fine scale or wrinkling, but although the consistency of the surface is changed, the lesion itself is not palpable.

Papule: A papule is a circumscribed, solid elevation of skin with no visible fluid, varying in size from a pinhead to less than either 5 or 10 mm in diameter at the widest point.

Plaque: A plaque has been described as a broad papule, or confluence of papules equal to or greater than 10 mm, or alternatively as an elevated, plateau-like lesion that is greater in its diameter than in its depth.

Nodule: A nodule is morphologically similar to a papule in that it is also a palpable spherical lesion less than 10 mm in diameter. However, it is differentiated by being centered deeper in the dermis or subcutis.

Tumour: Similar to a nodule but larger than 10 mm in diameter.

Vesicle: A vesicle is small blister, a circumscribed, fluid-containing, epidermal elevation generally considered less than either 5 or 10 mm in diameter at the widest point. The fluid is clear serous fluid.

Bulla: A bulla is a large blister, a rounded or irregularly shaped blister containing serous or seropurulent fluid, equal to or greater than either 5 or 10 mm, depending on one's definition of a vesicle.

Pustule: A pustule is a small elevation of the skin containing cloudy or purulent material (pus) usually consisting of necrotic inflammatory cells. These can be either white or red.

Cyst: A cyst is an epithelial-lined cavity containing liquid, semi-solid, or solid material.

Wheal: A wheal is a rounded or flat-topped, pale red papule or plaque that is characteristically evanescent, disappearing within 24 to 48 hours. The temporary raised bubble of taut skin on the site of a properly-delivered intradermal injection is also called a welt, with the ID injection process itself frequently referred to as simply "raising a wheal" in medical texts.

Telangiectasia: A telangiectasia represents an enlargement of superficial blood vessels to the point of being visible.

Burrow: A burrow appears as a slightly elevated, grayish, tortuous line in the skin, and is caused by burrowing organisms.

Secondary lesions

Scale: dry or greasy laminated masses of keratin that represent thickened stratum corneum.

Crust: dried sebum, pus, or blood usually mixed with epithelial and sometimes bacterial debris.

Lichenification: epidermal thickening characterized by visible and palpable thickening of the skin with accentuated skin markings.

Erosion: An erosion is a discontinuity of the skin exhibiting incomplete loss of the epidermis, a lesion that is moist, circumscribed, and usually depressed.

Excoriation: a punctate or linear abrasion produced by mechanical means (often scratching), usually involving only the epidermis, but commonly reaching the papillary dermis.

Ulcer: An ulcer is a discontinuity of the skin exhibiting complete loss of the epidermis and often portions of the dermis and even subcutaneous fat.

Fissure: A fissure is a crack in the skin that is usually narrow but deep.

Induration: dermal thickening causing the cutaneous surface to feel thicker and firmer.

Atrophy: refers to a loss of tissue, and can be epidermal, dermal, or subcutaneous. With epidermal atrophy, the skin appears thin, translucent, and wrinkled. Dermal or subcutaneous atrophy is represented by depression of the skin.

Maceration: softening and turning white of the skin due to being consistently wet.

Umbilication: formation of a depression at the top of a papule, vesicle, or pustule.

Phyma: A tubercle on any external part of the body, such as in phymatous rosacea.

# TOPIC 2.

## CLASSIFICATION OF DISEASES OF THE MUCOUS MEMBRANE OF THE ORAL CAVITY (MF DANILEVSKY, PT MAKSIMENKO, MKH-10).

**Oral Mucosal Diseases Listed According to Clinical Appearance**

| White Lesions | Red Lesions | Ulcerated Lesions | Vesiculobullous Lesions | Pigmented Lesions | Papillary/ Nodular Lesions |
|---|---|---|---|---|---|
| Frictional keratosis | Gingivitis | Traumatic ulcer | Benign mucous membrane pemphigoid | Amalgam tattoo | Papillary hyperplasia of the palate |
| Lichen planus (reticular and plaque forms) | Geographic tongue | Aphthous ulcer | Pemphigus vulgaris | Black hairy tongue | Fibroepithelial hyperplasia |
| Oral hairy leukoplakia | Median rhomboid glossitis | Syphilis (primary chancre) | Erythema multiforme | Oral Kaposi's sarcoma | Mucocele |
| Candidiasis (pseudo-membranous and hyperplastic forms) | Lichen planus (erosive form) | | Epidermolysis bullosa | Oral melanotic macule | Verruca vulgaris |
| Syphilis (mucous patches) | Benign mucous membrane pemphigoid<br>Pemphigus vulgaris<br>Erythema multiforme<br>Epidermolysis bullosa<br>Acute herpetic gingivostomatitis<br>Candidiasis (atrophic form)<br>Syphilis (mucous patches) | | Acute herpetic gingivostomatitis<br>Recurrent herpetic labialis<br>Herpes zoster<br>Herpangina | | Condyloma acuminatum<br>Focal epithelial hyperplasia |

## Systematics NMU (N.F. Danilevsky)

| Independently | Symptomatic | Syndromes |
|---|---|---|
| *Traumatic lesions* | | |
| Mechanical injury | | |
| Chemical injury | | |
| Physical trauma | | |
| Leukoplakia | | |
| *Infectious diseases* | | |
| Influenza virus, | measles, foot and mouth disease, chickenpox, infectious mononucleosis | AIDS |
| Bacterial | Whooping cough, diphtheria, scarlet fever, tuberculosis, syphilis, leprosy | Biedermann |
| Mycotic | | |

| | | |
|---|---|---|
| *Diseases of the mouth* | | |
| Eksfoliativny cheilitis | Eczematous cheilitis | |
| Meteorological cheilitis | Atopic cheilitis | |
| Actinic cheilitis | | |
| Chronic crack | | |
| Glandular cheilitis | | Puente Acevedo |
| Lymphedema | | Melkersson-Rosenthal, Miescher |
| | | |
| *Diseases of the tongue* | | |
| Desquamative glossitis | | Brock Potro |
| Folded tongue | | |
| Hairy tongue | | |
| Rhomboid glossitis | | |
| *Neoplasms* | | |
| Precancer | | |
| Benign tumors | | |
| Cancer and other malignant neoplasms | | |
| *In allergic lesions* | | |
| | Immediate reactions | Angioedema |
| | The reactions of delayed-type- purpura | Henoch |
| | Chronic recurrent aphthous stomatitis | |
| | Erythema multiforme | Stevens-Johnson, Lyell |
| *When dermatoses with an autoimmune component* | | |
| | Pemphigus | Dühring |
| | Pemphigoid | |
| | Cystic epidermolysis | |
| | Lichen planus | Greenspan |
| | Lupus erythematosus | |
| *When exogenous intoxications* | | |
| | Mercurial stomatitis | |
| | Lead stomatitis | |
| | Bismuth stomatitis | |
| *In diseases of the organs and systems* | | |
| | Alimentary canal | Rossolimo-spondylitis, Sebrella |
| | Cardiovascular system | Vesico-vascular, Rendu-Osler, Weber |
| | Endocrine | Addison, Cushing, Schmidt |
| | Nervous system | Glossodiniya |
| | Blood and blood-forming organs | Addison-Birmera, Vakeza, Verlgofa |
| | Hypovitaminosis | |
| | Collagenose | |

**The classification of diseases of the oral mucosa (P.T.Maksimenko)**

| **Primary** | | **Secondary (symptomatic)** | |
|---|---|---|---|
| *Traumatic* | | *When exogenous infections* | |
| Physical trauma | Manual<br>Thermal<br>Radiation<br>Electric | Bacterial | Scarlet fever, diphtheria, typhoid fever, whooping cough, gonorrhea, tuberculosis, syphilis, leprosy |
| Chemical injury | | Virus | Measles, influenza, shingles, chicken pox, foot and mouth disease, AIDS |
| *Autoinfektsionnye* | | *When noncommunicable diseases* | |
| Bacterial | Acute canker sores, necrotic stomatitis (gingivitis) | Gut gastro | Gastritis, colitis, peptic ulcer disease, hepatitis |
| Viral | Acute herpetic stomatitis, cheilitis<br>Recurrent herpetic stomatitis, cheilitis | Blood and blood-forming organs | Anemia, leukemia, agranulocytosis, hemorrhagic diathesis (illness thrombocytopenic purpura), polycythemia vera (a disease Vakeza) |
| Mycotic | Candida stomatitis, cheilitis, glossitis<br>Actinomycosis RBCU | Cardiovascular | Trophic ulcers, cystic syndrome and other |
| *Contact allergic (stomatitis, cheilitis, glossitis)* | | Radiation sickness | |
| | | Endocrine | Diabetes |
| | | Nervous system | Glossodiniya, xerostomia |
| | | Skin | Pemphigus<br>Lichen planus<br>Lupus erythematosus |
| | | Hypo-and avitaminosis | of Group B, C, A, E, PP |
| | | Heterointoxication | Mercury, lead, bismuth, difininovy gingivitis, stomatitis |
| | | Due to allergies | Stomatitis, glossitis, HRAS, Congenital syndromes, Stevens-Johnson syndrome |
| | | Congenital syndromes | Folded language, rhomboid glossitis, nevi and other |

Classification of Diseases MMOC ICD-10

By 12 Stomatitis and related lesions

Excludes: disintegrating oral ulcers (A69.0)

cheilitis (K13.0)

stomatitis (A69.0)

herpes (herpes simplex) gingivostomatit (V00.2)

noma (A69.0)

By 12.0 Recurrent oral aphthae

Aphthous stomatitis (large) (small)

Apthae Bednar

Recurrent muco-necrotic periadenit

Recurrent aphthous ulcers

Gerpetichny stomatitis

By 12.1 Other forms of stomatitis

Stomatitis:

• BDV

• dentition

• ulcerative

• vesicular

By 12.2 Cellulite and oral abscess

Cellulite mouth (bottom)

Abscess of submandibular region:

Excludes: Abscess:

• periapekalny (K04.6, K04.7)

• periodontal (K05.2)

• peritonsillar (J36)

• salivary gland (K11.3)

• Language (K14.0)

By 13 Other diseases of lip and oral mucosa

Included: damage lingual epithelium

Excludes: some damage to the gums and the toothless alveolar region (K05-K06)

cyst mouth (K09. -)

disease of language (K14. -)

Stomatitis and related lesions (K 12. -)

By 13.0 Diseases of lips

Cheilitis:

• BDV

• Angular

• exfoliative

• glandular

Heylodiniya

Cheilosis

Zayed NKIR

Excludes: ariboflavinoz (E53.0)

cheilitis due to radiation damage (L55-59)

Zayed due to:

• candidiasis (V37.8)

• lack of riboflavin (E53.0)

By 13.1 biting the cheeks and lips

By 13.2 leukoplakia and other lesions of the epithelium of the mouth, including the tongue

- Eritroplakiya oral epithelium
- Leykedema by language
- Nicotine leykokeratoz sky
- Sky smoker
- Excludes: hairy leukoplakia (K13.3)

By 13.3 hairy leukoplakia

By 13.4 granuloma and granulemopodobnye defeat of the oral mucosa

- Eozinofilnaya granuloma mucosal
- Pyogenic granuloma of the oral cavity
- Warty vitiligoidea

By 13.5 Sub mucosal fibrosis mouth

- Submucosal fibrosis language

By 13.6 Hyperplasia of the mucous membrane of the mouth due to irritation

- Excludes: hyperplasia toothless region (as a result of hyperplasia of dental prosthetics) due to irritation (K06.2)

By 13.7 Other and unspecified lesions of the mucous membranes of the mouth

- Focal mutsinoz mouth

By 14 Tongue Disorders

- Excludes: erythroplakia
- local tongue epithelial hyperplasia (K13.5)
- leykedemu
- leukoplakia

- hairy leukoplakia (K13.3)
- macroglossia (congenital) (Q38.2)
- submucosal fibrosis language (K13.5)

By 14.0 Glossitis

- Abscess language
- Ulcer (traumatic)
- Excludes: atrophic glossitis (K14.4)
- For 14.1 "geographic tongue"
- Benign migratory glossitis
- Eksfoliativny glossit

By 14.2 Median rhomboid glossitis

By 14.3 Hypertrophy of tongue papillae

- "Chorny hairy" tongue
- Coated tongue
- Hypertrophy of the leaf-buds
- Lingua villosa nigra

By 14.4 atrophy of tongue papillae

- Atrophic glossitis

By 14.5 Pleated language

- Split
- Borozchasty language
- Crinkly
- Excludes: split tongue, congenital (Q38.3)
- By 14.6 Glossodiniya
- Glossopiroz language
- Glossalgia

By 14.8 Other diseases language

- Atrophy
- Toothed language (s)
- Increased
- Hypertrophied
- By 14.9 The disease of language, unspecified
- Glossopatiya BDV

## TOPIC 3.

## FEATURES OF EXAMINATION OF PATIENTS WITH DISEASES OF ORAL MUCOUSE. PRIMARY AND SECONDARY LESION ELEMENTS

Oral examination commences with the visual examination of the lips and the vermilion border and by palpation after removing any lipstick. The lip is usually smooth and pliable. Maceration and cracking of the corners of the lips indicate angular cheilitis. Evert the lips and carefully inspect the labial mucosa . It should be smooth, soft and well-lubricated by minor salivary glands that can be palpated. One may observe a in the lower lip resulting from trauma to the minor salivary gland ducts, as the lower lip is frequently prone to injury, mucocele particularly from accidental biting.

The buccal mucosa is examined by stretching it with a pair of tongue depressors or mouth mirrors after the subject partially opens the mouth). In people with dark skin, one may frequently observe a benign condition called, which is characterized by a diffuse greyish white opalescence in the buccal mucosa; this leukoedema disappears when the tissue is stretched.

A horizontal white or grey line, along the buccal mucosa, called_may be

observed in some persons (This is a benign, hyperplastic reaction resulting from the chronic irritation from the teeth cusps at the level of the interdigitation of the teeth. The opening of the parotid salivary gland duct, the Stensen duct, may be observed as a small papillary or punctate soft tissue mass on the buccal mucosa adjacent to the maxillary second molar tooth. Milking of the parotid gland may expel saliva at the duct opening. Ectopic sebaceous glands may be observed on the buccal or labial mucosa as whitish-yellow, pinpoint papules; this developmental anomaly is termed as Fordyce conditions or granules. Minor salivary glands and_may lead to a granular feel on palpation of the buccal mucosa.

After examination of the buccal mucosa, the dorsal surface of the tongue is examined by asking the subject to protrude the tongue and attempt to touch the tip of the chin); alternatively the tip of the tongue may be held gently by the fingers and a gauze sponge sensation.

Other tests might include: swabs, both bacterial and viral tissue scrapings or swabs for fungal infections biopsy or the removal of cells or tissue for further study blood tests patch tests to identify allergy.

## TOPIC 4.

## TRAUMATIC LESIONS OF THE ORAL MUCOSA (MECHANICAL, CHEMICAL, PHYSICAL, ELECTRICAL INJURY). ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSIS, TREATMENT AND PREVENTION.

Chronic mechanical injury (trauma mechanicum chronicum)

Complaints:

sensation of discomfort, pain, swelling; frequent biting;

presence of the old ulcer; growth in the hard palate, the gums, the tongue, under the prosthesis; whitish section of the mucous membrane in the place of the permanent trau matic factor.

Lymph nodes are increased, painful during palpation. The manifestation of changes depends on gravity of the lesion.

Inflammatory spot or erythema in the place of the injury. • Localization of erosion corresponds to the traumatic agent, erosion is painful, localized on the hyperemized mucous membrane. • Indolent ulcer is localized more frequently on the tongue, lips, cheeks along the line of teeth joining, and also in the limits of orthopaedic field. It is sin- gle, painful, surrounded with inflammatory infiltration, the bottom is most often uneven, covered with fibrinous fur. The decrease, the disappearance of painfulness, the appearance of papillary growths indicate about malignization.

Clinic:

Papillomatous hyperplasia – papilloms with the soft, grainy, bright red sur- face under the prosthesis, they are more frequent localized in the region of the hard palate.

• With the habit to bite or to suck lips, tongue, and cheeks mucosa membrane (in essence along the line of teeth joining) acquires the unique form: it will swell, it has the white macerated surface in the form of either spots and large illegibly limited sections, or fringed form because of many small patches of the unevenly biting epithelium. Lesion has asymptomatic course, but during the deep removal the erosions are formed, they are painful in contact with chemical stimuli.

Treatment:

The elimination of the traumatic agent.

Anesthetization.

Cleaning the surface of erosion and ulcer from the necrotic fur.

Processing of ulcer and oral cavity by the solutions of the antiseptics.

Stimulation of the epithelisation.

Physical and mechanical traumas of oral mucosa

Linea alba (white line)

Localization: Buccal mucosa, at the level of the occlusal line of the teeth. It is a horizontal streak on the buccal mucosa at the level of the occlusal plane extending from the commissure to the posterior teeth.

Clinical features: Lesions are mostly asymptomatic. The common visual symptom of linea alba is the presence of whitish, linear, filament-like plicae formations, horizontally parallel to the occlusal level of bicuspids and molar teeth in both left and right sides of buccal mucosa. Palpation should give a tactile sensation of normal mucosa texture. It is more prominent in individuals with reduced overjet of the posterior teeth. It is often scalloped and restricted to dentulous areas. The diagnosis is based on clinical grounds alone.

Etiology: Lesions mainly arise from occlusal traumas of posterior teeth generated due to the parafunctional cheek sucking of patient. The sucking habit is also associated with friction between buccal tubercules and irritates the buccal mucosa by pressure. Prevalence of such lesions is about 6.2–13% in the population.

Treatment: No treatment is required; the white streak may disappear spontaneously in some people. But very sharp-edged teeth can be corrected.

Chronic biting (Morsicatio buccarum)

Localization: The lesions made by chronic bite trauma (nibbling) on the buccal mucosa generally cause keratinized shreds, tissue tags, or erosive and desquamative surfaces. These lesions according to their localizations are called as “morsicatio buccarum” if they are localized on the buccal mucosa, “morsicatio labiorum” if they appear on the labial mucosa, and “morsicatio linguarum” if they occur on the lateral borders of the tongue. The lesions are seen on the buccal mucosa, bilateral chewing line, labial mucosa, and lateral edges of the tongue.

Clinical features: Lesions are apparent as shallow whitish wrinkles which are diffuse and present irregularly on the buccal, labial mucosa, and tongue. Epithelial desquamation occurs on the surface. In some cases, erosions and petechiae may be seen. The lesions could be diagnosed by clinical inspection.

Etiology: It is often related to chronic biting of the oral mucosa seen in psychologically tense patients. Parafunctional bite of the buccal mucosa, lips, and tongue until wear of superficial epithelium and wound formation is consciously made by those patients. The incidence of morsicatio buccarum was reported to be 2.5% in Caucasian populations.

Treatment: Treatment is usually unnecessary. It is recommended to stop the habit. Psychological treatment can be suggested for stopping a bad habit. Acrylic splint can be made on the occlusal surface of the teeth. It is accepted as a precancerous lesion.

Traumatic ulcers

Localization: Presence of traumatic ulcers is a relatively common finding in dental practice. Such lesions arise from trauma related to bite of buccal mucosa, lateral border of the tongue or lips during chewing. Traumatic ulcers seen in the mucobuccal folds and gingiva are related to different irritant factors such as hard foods and inappropriate hard brushing. Traumatic ulcer due to lip biting after inferior dental nerve block is seen on the lower lip. During orthodontic treatment, traumatic ulcers can occur especially on the buccal mucosa due to the irritation of braces or appliance wires.

Clinical features: Traumatic ulcers could be of solitary shallow or deep discontinuity type showing on the epithelium and are associated with peripheral keratosis of mild to severe degree. The bottom of the ulcerative lesions is made of whitish or yellow pseudomembrane. Upon elimination of the causative factor, often the ulcer heals with or without scar depending on the extent of the damage occurred.

Etiology: They could originate from accidental mucosal biting, sharp edges of prosthesis, sharp or pointed food stuff, during orthodontic treatment, lip biting after injection of local anesthetic solutions, neonatal teeth, or faulty tooth brushing. During dental treatments, iatrogenic damages can result in traumatic ulcer formation. Some medical treatments can cause oral ulcerations, such as brutal intubation for general anesthesia, ENT surgeries, or endoscopic interventions and iatrogenic malpractice applications. A high prevalence of traumatic ulcer of about 21.5% was reported among lower classes of Brazilian population. Most prevalent types of lesions were reported to be traumatic ulcer and actinic cheilitis (7.5% for each). Among the etiological factors of traumatic ulcers could be mentioned traumas caused by bites, dental appliances, inappropriate tooth brushing, misfit of removable partial or total dentures, irritating caries edges, malocclusion and puncturing restorations.

Treatment: Most often, traumatic ulcers can heal spontaneously and uneventfully without complications in a brief period of time. But, in case of persistent traumatic factors, such as presence of sharp tooth morphology, cutting edges of restorations, and puncturing appliance contours, especially inadequate surfaces of removable prosthesis, continuous trauma arising from above-mentioned causes can lead to formation of chronic ulcers.

Chemical injuries of the oral mucosa

Chemical burn

Localization: Gingiva and mucobuccal folds are main localization regions of such lesions.

Clinical features: The wounds have irregular shape and white color, are overlaid by a pseudomembrane, and are very painful. Lesions can cover an extended area. If the lesions are contacted shortly, a shallow whitish and wrinkled appearance occurs. Brief contacts cannot cause necrosis.

Etiology: Caustic chemical and drug materials when they come in contact with the oral mucosa are often very irritating and cause direct mucosal trauma. Inappropriate usage of medications, such as aspirin application onto the neighboring mucosa of painful teeth with decay, may result in mucosal trauma. Iatrogenically, during dental treatments irrigant solutions (sodium hypochlorite or formalin) or some endodontic pastes with arsenic can irritate the mucosa. However, such injuries are not very common since the introduction of rubber dam in dental practice.

Treatment: The best treatment of chemical burns of the oral cavity is prevention. The proper use of a rubber dam during endodontic procedures reduces the risk of iatrogenic chemical burns. Superficial burns of mucosa can heal in a short period of time (within 1 or 2 weeks) as the turn-over of oral mucosa is very high. Oral surgery and antibiotics are necessary in very rare cases. Gel with hyaluronic acid can accelerate the healing process. Possible treatments after chemical injuries, in relation with the severity of wounds, are topical and intralesional corticosteroid applications, caustic acid ingestion, commissuroplasty, mucosal flaps, free radial forearm flap and free jejunal graft, surgeries made with electrocautery or soft tissue laser, and wound coverage by periodontal pack

Contact allergic stomatitis

Localization: Contact area of oral mucosa due to denture base materials, restorative materials, mouthwashes, dentifrices, chewing gums, food, and other substances. Various chemical or natural agents in contact with the mucosa can irritate and cause contact stomatitis. For example, cinnamaldehyde or cinnamon essential oil, which are commonly used as flavoring agents in foods, beverages, candies, and hygiene products by contact with mucosal surfaces, may trigger the formation of allergic stomatitis.

Clinical features: Diffuse erythema, edema, occasionally small vesicles, and shallow erosions appear immediately after contact with the allergen on the affected mucosal surfaces. Lesions are associated with burning symptom. In chronic allergies, whitish, hyperkeratotic, erythematous lesions form .

Etiology: Denture base materials, restorative materials like amalgam, mouthwashes, dentifrices, chewing gums, food, and other substances may be responsible.

Treatment: Contact allergic stomatitis can be diagnosed by an accurate examination and clear understanding of medical history of the patient. Clinician's diagnostic ability and experience are highly important to avoid further unnecessary examinations, invasive and expensive diagnostic procedures. Treatments include removal of suspected allergens, and use of topical or systemic corticosteroids, antihistamines.

Radiation injuries

Oral mucositis

Localization: Developments in oncology have led to improved survival rates for different cancers. Unfortunately, those treatment regimens have side effects such as formation of oral mucosal lesions. The most common wound type during chemotherapy is oral mucositis which appears by inflamed erosive or ulcerative lesions on mucosal surfaces in the oral cavity. Generally, buccal mucosa is affected by radiation treatment of head and neck tumors.

Clinical features: After radiotheraphy, at the end of first week, the first oral manifestations can appear. Lesions are erythematous and edematous. In the following days, ulcerative erosions with whitish-yellow exuadate appear. As salivary glands are involved, xerostomia occurs and is followed by tongue papillary changes with loss of taste, burning sensation, and pain during function. Speech is also affected negatively.

Etiology: Chemotherapy, radiotherapy, or their combinations can lead oral mucositis. The majority of patients (approximately 20–40%) receiving conventional chemotherapy regimens for solid tumors, in relation to the dose and cytotoxicity of the drug used, have oral mucositis. It is a side effect of radiation treatment of head and neck tumors.

Treatment: Supportive care, cessation of radiation treatment, B-complex vitamins, and sometimes low doses of corticosteroids are suggested.

Electrical and thermal burn

Electrical burn

Localization: Commissures of the mouth often result in severe facial scarring and deformation. Most commissural electrical burns involve mucosa, submucosa, muscle, nerve, and vascular tissue.

Clinical features: Damage made accidentally to lingual or/and labial arteries can cause abundant bleeding. When burned tissues spontaneously start to loosen or slough and occasional trauma occurs, this type of bleeding happens. Generally, this is observed 3–4 days after burn injury. Pressure should be applied to the hemorrhage site to stop the bleeding and the patient should be taken to the nearest hospital emergency room for definitive care.

Etiology: The majority of electrical burns are home accidents. Generally, children play with live electric extension cables/cords and may contact or suck them and are injured by current. Especially in the cable/plug junctions, in non-fitted appliance plugs, the electric current flows

through tongue or oral cavity when they are in contact with saliva, and the electric energy burns oral tissues. Children under three years of age are mostly affected by this type of injury.

Treatment: Whatever is the severity of burned injury, the basic treatment strategy involves pain relief, infection control, and acceleration of wound repair. Application of antibiotic ointments to the burn area has been recommended by some authors. Systemic antibiotics are recommended by most clinicians to prevent wound infection. Facial disfigurement takes place if splints are not applied. Microstomia reduces mouth opening, renders oral hygiene difficult, and decreases functions of speech and chewing. Most of the cases need plastic surgery.

Thermal burn

Localization: Oral mucosa, especially tongue and palatal mucosa.

Clinical features: Clinically, the condition appears as a red or white, painful erythema that may undergo desquamation, leaving erosions. In excessive damage to tissues, necrosis could appear. In mild lesions, wounds can heal spontaneously within a week.

Etiology: Thermal burns mostly happen by accidental ingestion of hot substances. High incidence of thermal burns with a prevalence of 24.6% is seen among children and young patients. Usually caused due to contact with very hot foods, liquids, hot metal objects or iatrogenic usage of lasers (diodes, Nd:YAG, Er:YAG or CO2), piezoelectric surgery, or electrosurgery devices.

Treatment: No treatment is required for simple lesions. Care should be taken in deep lesions to avoid contamination during healing period. Saline would be prescribed to accelerate wound healing and avoid bacterial ingrowth. Ozone therapy and laser biomodulation could help for good prognosis. In severe damages, prophylactic antibiotic coverage is recommended. In hard tissue damages related to thermal burn, the necrotic area should be removed surgically in order to avoid damage to surrounding vital tissues and obtain blood supply for repair and subsequent regeneration.

## TOPIC 5.

## PRIMARY AUTOINFECTIVE STOMATITIS. ACUTE CATARRHAL STOMATITIS. ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSIS, TREATMENT, PREVENTION. ACUTE HERPETIC STOMATITIS. ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSIS, TREATMENT, PREVENTION.

Stomatitis is inflammation of the mouth. It affects the mucous membranes

Stomatitis is a type of mucositis, a condition defined as pain or inflammation of the mucous membrane.

Mucositis is a relatively common side effect of chemotherapy and sometimes radiotherapy. It can affect the inside of the lips, cheeks, gums, tongue, and throat.

There are two main types of stomatitis: Canker sores

These are also known as aphthous ulcers and are part of the most common cause of stomatitis. The sores are pale white or yellowish in color with a red outer ring.Canker sores can develop singly or in a cluster and usually occur on the inside of the lips or cheek, or on the tongue.

Cold sores Cold sores are small, painful, fluid-filled sores that usually occur on or around the lips near the edge of the mouth. Caused by the herpes virus (HSV), the condition is also known as herpes stomatitis.Stomatitis can be broken down into different categories, depending on which area of the mouth is affected: cheilitis – inflammation of the lips and around the mouth glossitis – inflammation of the tongue gingivitis – inflammation of the gums pharyngitis – inflammation of the back of the mouth The most common causes are: trauma from ill-fitting dentures or braces, biting the inside of the cheek, tongue, or lip, and surgery chemotherapy treatment for cancer viral infection, such as herpes yeast infection, such as thrush any condition associated with xerostomia, or dry mouth smoking or chewing tobacco

Other examples include: bacterial infections sexually transmitted infections weakened or deficient immune system irritation from strong chemicals stress certain diseases, including Behcet's disease, Crohn's disease, and lupus medications, including sulfa drugs, anti-epileptics, and some antibiotics nutritional deficiencies allergic reactions burns caused by hot food and drink It is important to identify the cause of stomatitis in order to treat it properly.

Stomatitis often results in pain, stinging, and soreness. Each person may experience different symptoms. These can include mouth ulcers with a white or yellow layer and red base, usually inside the lips, cheek, or on the tongue red patches blisters swelling oral dysaesthesia a burning feeling in the mouth lesions that heal in 4-14 days and often recur.

Diagnosis will depend entirely on what is causing the stomatitis. Relevant investigations include a physical examination, as doctors can learn a lot by looking at the appearance and distribution of ulcers.

## TOPIC 6.

## ACUTE APHTHOUS STOMATITIS. ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSIS, TREATMENT, PREVENTION.

Aphthous stomatitis (RAS) is a common condition affecting the oral mucosa, as it has been estimated that 20% of the general population suffers from it at some time in life. It is characterized by sharply demarcated, round to oval ulcers with an erythematous halo.

The ulcers heal without treatment after one to several weeks, depending on the subtype of RAS (see below). In some individuals, the ulcers are preceded by a recognizable prodromal stage that has been described variously as a tingling sensation, mucosal edema, or the appear ance of small nodules. The ulcers are typically painful, and during severe attacks of RAS, eating and speaking become greatly impaired.

Three forms of RAS have been described: major, minor, and herpetiform. Minor RAS is the most common form; ulcers do not exceed 1 cm in diameter and heal without scarring in seven to 10 days. The ulcers of major RAS are larger, deeper, and more numerous. They heal with scarring. In herpetiform RAS, crops of pinhead-size ulcers appear, and each crop of ulcers lasts seven to 10 days. As the name implies, these ulcers bear some clinical resemblance to herpetic lesions, but herpes virus cannot be cultured or demonstrated in them, and they are generally thought to represent a variant of RAS.

The etiology of RAS remains unknown, although several -hypotheses have been proposed, including hypersensitivity to an L-form streptococcus and a local immune response to antigenically altered mucosa. Aphthous ulcers are usually diagnosed without much difficulty on the basis of their characteristic clinical appearance and history of previous self-limited ulcers.

Many forms of treatment have been tried to alleviate pain and promote healing of the ulcers, but none has been completely effective. Tetracycline Geographic Tongue mouthrinses are often helpful, especially if used during the early phase of ulceration. Occlusive ointments such as Orabase, used alone or with topical steroids, have also been reported to reduce symptoms. In most patients suffering from RAS, the ulcers are not accompanied by extra-oral manifestations.

Recurrent selflimited ulcers that are clinically indistinguishable from aphthous ulcers may occur in patients with inflammatory bowel disease; in these patients, the course of the oral ulcers closely parallels that of exacerbations of the bowel disease. Aphthous ulcers may also be seen as part of Behcet's syndrome, a systemic inflammatory disease of unknown etiology that most commonly affects young male adults. Behcet's disease is characterized by the triad of oral ulcers, genital ulcers, and ocular inflammation. In addition, the inflammatory changes of this disease are now known to affect multiple tissues and organs, including skin, joints, blood vessels, heart, lungs, and the central nervous system.

## TOPIC 7.

## CHRONIC RECURRENT HERPES. CAUSES, PATHOGENESIS, CLINIC, DIAGNOSIS, TREATMENT, PREVENTION.

Recurrent herpes simplex labialis, also known as oral herpes, is a condition of the mouth area caused by the herpes simplex virus. It's a common and contagious condition that spreads easily.

Herpes simplex labialis is the result of a virus called herpes simplex virus type 1 (HSV-1). The initial acquisition usually occurs before age 20. It typically affects the lips and areas around the mouth.

You can get the virus from close personal contact, such as through kissing, with someone who has the virus. You can also get oral herpes from touching objects where the virus may be present. These include towels, utensils, razors for shaving, and other shared items.

Events that trigger a recurrence of oral herpes might include: fever menstruation a high-stress event fatigue hormonal changes upper respiratory infection extreme temperature a weakened immune system recent dental work or surgery.

The original acquisition may not cause symptoms at all. If it does, blisters may appear near or on the mouth within 1 to 3 weeks after your first contact with the virus. The blisters might last up to 3 weeks. In general, a recurrent episode is milder than the initial outbreak.

Symptoms of a recurrent episode may include:

blisters or sores on the mouth, lips, tongue, nose, or gums

burning pain around the blisters

tingling or itching near the lips

outbreaks of several small blisters that grow together and may be red and inflamed treatment oral antiviral medicines to fight the virus, such as: acyclovir famciclovir -valacyclovir.

The chronic form is manifested by the clinical symptoms of the disease for a long time. A delayed infection is characterized by a long incubation period (years), a slow course with the development of severe clinical symptoms. The chronic form of persistence of the virus in the oral cavity is recurrent herpes simplex (Herpes chronica recidiva) of the oral mucosa (chronic recurrent herpes, recurrent herpetic stomatitis, gingivostomatitis), recurrent herpes of the lips and herpetic recurrent ganglioneuritis. In rare cases, there are recurrent herpetic lesions of the esophagus,

pharynx and larynx. Recurrent herpes simplex is detected mainly in adults and every tenth child (12.5%) who has suffered from acute herpetic stomatitis. The provoking factors of relapse can be acute infectious diseases, pneumonia, exacerbation of chronic diseases of the digestive system, exacerbation of dental foci of infection and periodontal diseases, ENT diseases, hypothermia, the effect of radiation, insolation, vitamin deficiencies, stress, trauma, surgical interventions, dental manipulations (injection, separation, preparation, matrix imposition), taking medications that suppress immune defenses, and the like. Under the influence of these factors, the virus, which is in the body in a latent phase, can become active and cause a relapse. More often women are affected (a clear connection with the menstrual cycle). For herpetic lesions, an increase in the incidence in the autumnwinter period is characteristic (in approximately 75% of cases, the occurrence of a relapse was preceded by severe hypothermia). The development of the disease is evidence of a decrease in general immunity and reactivity of the oral mucosa, it can manifest itself on the skin (Herpes simplex labialis, nasalis recidiva) and mucous membranes (Stomatitis herpetica recidiva). The clinical manifestations of both primary herpes infection and recurrent forms can be the same. Recurrent herpes of the oral mucosa is more often localized on the red border of the lips, on the border with the skin, hard palate, tongue, gums and occurs mostly as a result of the action of the above provoking factors. Aggravation begins with the appearance of various kinds of subjective sensations - "harbingers of relapse". They occur on average a day before the appearance of rashes on the corresponding area of the skin or mucous membranes and manifest themselves in the form of itching, burning, rarely pain. Subsequently, a lesion is formed there. In this area, erythema occurs, the size of which and the intensity of the color can vary. The development of erythema is accompanied by moderate edema. On the erythematous-edematous background, round bubbles form. The bubbles are arranged in groups, closely adjacent to each other, sometimes merge and form larger bubbles with scalloped edges. Isolated small bubbles can be found along their periphery. The contents of the bubbles are first transparent, then cloudy. Bubbles exist for 2-5 days. Subsequently, they rupture, forming erosion with polycyclic edges, which are covered with crusts or bloom. In the first few days after the onset of erosions, they are very painful, while a painful reaction of regional lymph nodes is observed. Crusts can also form as a result of drying bubbles that have not opened. After 7-9 days, the crusts are rejected, after which a slightly pink or hyperemic spot remains, which disappears after 1-2 weeks. The general condition of patients with recurrent herpes simplex of the mucous membrane of the mouth and lips is usually not disturbed; in young people, intoxication and different temperature reactions are possible. Depending on the frequency of relapses, mild (1-2 relapses within 3 years), moderate (1-2 relapses per year) and severe (4-5 relapses per year or permanent course) forms of recurrent herpes are distinguished. Less often, recurrent herpes simplex is characterized by atypical clinical forms, in the symptomatology of which one of the components of inflammation, one of the stages of the inflammatory process, or subjective symptoms prevail. This predetermines the corresponding name of the form: abortive (itching, erythematous, papular), edematous (elephantiazoid), zosteroid, hemorrhagic, erosive-ulcerative, necrotic, rupioid, burn. The diagnosis is made on the basis of the clinical picture, cytological examination (detection of herpes cells) and laboratory methods; enzyme immunoassay (ELISA) and polymerase chain reaction (PCR). During a histological examination of the drug, ballooning dystrophy is determined, which is manifested by focal changes in the cells of the oste-like layer, which take the form of balls, are separated from one another. At the same time, as a result of amitotic division, multinucleated large cells are formed. Serous exudate separates the altered cells, forming a vesicle cavity filled with exudate with epithelial cells. An acute inflammatory process is observed in the adjacent lamina propria of the oral mucosa. Treatment. In order to treat viral lesions of the oral mucosa, the following measures should be applied: 1) neutralize the virus (etiotropic antiviral therapy) and prevent new rashes of lesion elements; 2) eliminate signs of general intoxication; 3) to enhance the level of immunological resistance of the

body and the oral mucosa; 4) eliminate pain, accelerate the cleansing of erosion, reverse development of the inflammatory reaction and epithelialization of the lesion elements. However, the treatment that is carried out during acute manifestations of herpes infection and its recurrence, unfortunately, is not a guarantee that a new relapse of the disease will not develop. To prevent it, it is necessary to carry out a whole range of therapeutic measures. As an example, the program of I.O. Isakov, 1991. Principles of staged treatment and prevention of herpes infection Stage I - treatment in the acute period of the disease (relapse): 1. Antiherpetic drugs (topically, orally, intravenously). In persons with immunodeficiency, there is a mandatory double dose. 2. Appointment of antioxidants (tocopherol acetate, etc.), course - 14 days. 3. With significant exudation from the elements of the lesion, the use of prostaglandins (indomethacin, etc.) for 14 days is indicated. 4. Antiviral (etiotropic) drugs with different mechanisms of action in combination with immunomodulators. Stage II - therapy during remission (early convalescence): 1. Immunomodulators (can be the same as in the acute period). 2. Adaptogens of plant origin. 3. In case of severe immunosuppression, a short course of thymus preparations. Stage III - specific prevention of recurrence of herpes infection using herpes vaccines. The goal of vaccination is to activate the patient's cellular immunity, its immunocorrection and specific desensitization of the organism until a stable clinical and immunological remission is achieved. Stage IV - dispensary observation and rehabilitation of patients with herpes infection. Carrying out therapeutic measures of this comprehensive program and dispensary observation of patients with herpes infection makes it possible to significantly reduce the risk of relapse of the disease.

## TOPIC 8.

## ACUTE ULCERATIVE STOMATITIS. ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSIS, TREATMENT, PREVENTION.

Acute necrotizing ulcerative gingivitis (ANUG) is an endogenous oral infection that is characterized by necrosis of the gingiva. Occasionally, ulcers of the oral muco- sa also occur in patients with hematologic disease or severe nutritional deficiencies. Studies have shown that the disease is accompanied by an overgrowth of organ- isms prevalent in normal oral flora and is not transmissible. The organisms most frequently mentioned as working symbiotically to cause the lesions are the fusiform bacillus and spirochetes. Plaque samples taken from ANUG patients demonstrate a constant anaerobic flora of Treponema spp, Selenomonas spp. Fusohacterium spp, and Bacteroides intermedius. The tissue destruction is thought to be caused by en- dotoxins that act either directly on the tissues or indirectly by triggering immunologic and inflammatory reactions.

Classic ANUG in patients without an underlying medical disorder is found most often in those between the ages of 16 and 30 years, and it is associated with three major factors: 1. Poor oral hygiene with pre-existing marginal gingivitis or faulty dental res- Acute necrotizing ulcerative gingivitis torations. 2. Smoking 3. Emotional stress. Systemic disorders associated with ANUG are diseases affecting neutrophils (such as leukemia or aplastic anemia), marked malnutrition, and HIV infection. Malnutrition-associated cases are reported from emergent countries where the un- treated disease may progress to noma, a large necrotic ulcer extending from the oral mucosa through the facial soft tissues.

A fulminating form of ulcerative stomatitis related to ANUG is noma (cancrum oris), which predominantly affects children in sub-Saharan Africa. This disease is characterized by extensive necrosis that begins on the gingiva and then progress- from the mouth through the cheek to the facial skin, causing extensive disfigure- ment. The major risk factors associated with noma include malnutrition, poor oral hygiene, and concomitant infectious diseases such as measles. Living in close prox- imity to livestock is also believed to play a role, and Fusobacterium necrophorum, pathogen associated with disease in livestock, has been isolated from over 85 % noma lesions. The mortality rate without appropriate therapy exceeds 70 %.

Clinical manifestations. The onset of acute forms of ANUG is usually sudden, with pain, tenderness, profuse salivation, a peculiar metallic taste, and spontane- cus bleeding from the gingival tissues. The patient commonly experiences a loss of the sense of taste and a diminished pleasure from smoking. The teeth are fre- quently thought to be slightly extruded, sensitive to pressure, or to have a "woody sensation." At times they are slightly movable. The signs noted most frequently are gingival bleeding and blunting of the interdental papillae. The typical lesions of ANUG consist of necrotic punchedout ulcerations, developing most commonly on the interdental papillae and the marginal gingivae.

These ulcerations can be ob- served most easily on the interdental papillae, but ulceration may develop on the cheeks, the lips, and the tongue, where these tissues come in contact with the gin- gival lesions or following trauma. Ulcerations also may be found on the palate and in the pharyngeal area. When the lesions have spread beyond the gingivae, blood dyscrasias and immunodeficiency should be ruled out by ordering appropriate labo- ratory tests, depending upon associated signs and symptoms. The ulcerative lesions may progress to involve the alveolar process, with se- questration of the teeth and bone.

When gingival hemorrhage is a prominent symp- tom, the teeth may become superficially stained a brown color, and the mouth odor is extremely offensive. The tonsils should always be

examined since these organs may be affected. The regional lymph nodes usually are slightly enlarged, but occa- sionally the lymphadenopathy may be marked, particularly in children. The constitutional symptoms in primary ANUG are usually of minor signifi- cance when compared with the severity of the oral lesions. Significant temperature elevation is unusual, even in severe cases, and, when it exists, other accompanying or underlying diseases should be ruled out, particularly blood dyscrasias and AIDS. HIV-infected patients with NUG have rapidly progressing necrosis and ulceration first involving the gingiva alone, and then NUP with the periodontal attachment and nvolved alveolar bone. The ulcerated areas may be localized or generalized and often are very painful. In severe cases, the underlying bone is denuded and may become sequestrated, and the necrosis may spread from the gingiva to other oral tissues found Treatment.

The therapy of ANUG uncomplicated by other oral lesions or sys- temic disease is local débridement. At the initial visit, the gingivae should be dé- brided with both irrigation and periodontal curettage. The extent of the débridement depends on the soreness of the gingivae. The clinician should remember that the more quickly the local factors are removed, the faster is the resolution of the lesions. Special care should be taken by the clinician to débride the area just below the marginal gingivae.

## TOPIC 9.

## CHRONIC ULCERATIVE STOMATITIS. ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSIS, TREATMENT, PREVENTION.

Chronic ulcerative a rare autoimmune disease that presents as painful ulcers in the mouth, often coming and going for many years.

Unlike other autoimmune diseases that cause mouth blisters and ulcers, it does not respond well to corticosteroids. Stomatitis means a sore mouth.

Chronic ulcerative stomatitis is a rare disease with less than 100 cases so far reported. It seems to almost exclusively affect white women in late middle age with an average age at onset of 60 years. There are rare reports of chronic ulcerative stomatitis affecting males or younger women.

Clinical features presents as painful erosions (superficial ulcers) in the mouth which can resemble erosive lichen planus. It may mimic other autoimmune conditions that cause mouth ulcers or erosions including pemphigus vulgaris and cicatricial pemphigoid, or resemble a desquamative gingivitis. The ulcers can occur on the gums, inside the cheeks or on the tongue. The condition lasts many years, sometimes coming and going. Very rarely similar lesions have also been reported on the genital or conjunctival mucosa. Skin lesions resembling lichen planus have been reported in some patients. It is not yet clear if chronic ulcerative stomatitis coexists with lichen planus or is a variant of it. The patient is otherwise well.

Mucosal biopsies from the mouth show nonspecific pathological features, most often resembling lichen planus direct immunofluorescence (DIF) Indirect immunofluorescence (IIF).

An ELISA test assay has recently been developed to detect the specific autoantibodies. Major criteria erosive or exfoliative lesions in the mouth characteristic indirect and direct immunofluorescence (IIF and DIF) Minor criteria chronic course with relapses female in the older age group response to hydroxychloroquine alone or combined with small doses of cortisone.

Treatment of chronic ulcerative stomatitis. Chronic ulcerative stomatitis typically does not respond to topical steroids or oral steroid treatment. However there is usually an excellent initial response to oral hydroxychloroquine, 200-400mg/day. This may result in a longlasting remission. However in the longterm, hydroxychloroquine may not prevent relapses and low dose systemic corticosteroids &/or dapsone may be required in addition.

## TOPIC 10.

## FUNGAL LESIONS OF THE ORAL MUCOSA. ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSIS, TREATMENT AND PREVENTION.

Oral thrush happens when a yeast infection develops inside your mouth. It's also known as oral candidiasis, oropharyngeal candidiasis, or simply thrush.Oral thrush most often occurs in infants and toddlers. It causes white or yellowish bumps to form on the inner cheeks and tongue. Those bumps usually go away with treatment.The infection is typically mild and rarely causes serious problems. But in people with weakened immune systems, it can spread to other parts of the body and cause potentially serious complications

Symptoms of oral thrush

In its early stages, oral thrush may not cause any symptoms. But as the infection gets worse, one or more of the following symptoms may develop:

white or yellow patches of bumps on your inner cheeks, tongue, tonsils, gums, or lips

slight bleeding if the bumps are scraped

soreness or burning in your mouth

a cotton-like sensation in your mouth

dry, cracked skin at the corners of your mouth

difficulty swallowing

a bad taste in your mouth

a loss of taste

Causes of oral thrush

Oral thrush and other yeast infections are caused by an overgrowth of the fungus Candida albicans (C. albicans).

Treatment for oral thrush

To treat oral thrush, your doctor may prescribe one or more of the following medications:

fluconazole (Diflucan), an oral antifungal medication clotrimazole (Mycelex Troche), an antifungal medication(Nystop, Nyata), an antifungal mouthwash that you can swish in your mouth or swab in your baby's mouth

## TOPIC 11.

## FLU. MEASLES. ETIOLOGY, PATHOGENESIS, CLINICAL MANIFESTATIONS OF THE ORAL MUCOSA, DIAGNOSIS, TREATMENT AND PREVENTION.

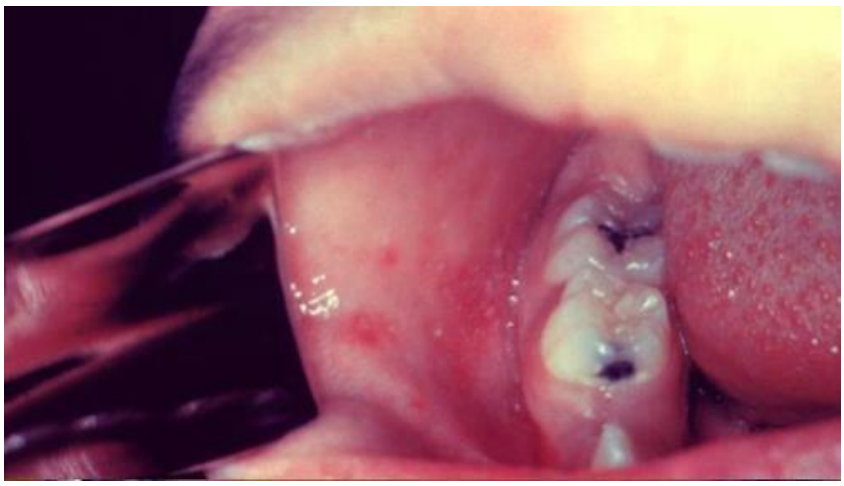
FLU (grippe) is an acute contagious respiratory disease caused by different types of influenza viruses. Etiology, pathogenesis. Infection occurs by airborne droplets from direct contact with a sick person, which is a source of infection from the last hours of the incubation period to 4-7 days of illness. Influenza is characterized by seasonality (winter-spring), the course in the form of epidemics. The rapid spread of influenza is due to the ease of airborne infection, the variability of the antigenic structure of the influenza virus, and a short incubation period. Influenza pathogens belong to the family of orthomyxoviruses, which include three antigenic types - A, B, C. They quickly die from heating, drying, under the influence of disinfectants and ultraviolet radiation. Influenza viruses have enzymatic, toxic, infectious and antigenic activity. Infection spreads to the upper respiratory tract. Viruses damage the mucous membrane of the respiratory tract. Degenerative changes in the cylindrical epithelium occur, accompanied by a decrease in the barrier function of the mucous membrane. Influenza viruses are able to adsorb on special red blood cell receptors, which lead to their agglutination. The penetration of the virus and its toxins into the blood causes an increase in the permeability and fragility of the vascular walls (the development of hemorrhagic syndrome), toxic damage to various parts of the central and autonomic nervous system. Vagal innervations predominates (increased secretion of mucus in the bronchi, bradycardia, decrease of blood pressure). Influenza infection causes the appearance of immunodeficiency, which leads to secondary bacterial complications and exacerbation of concomitant chronic diseases. But from the first hours, interferon is formed in infected cells, which limits the spread of the influenza virus. By the end of the week of illness, titers of humoral antibodies appear and increase. Antibodies (secretory immunity) also circulate in the secrets of the mucous membranes of the respiratory tract. The severity of the disease is determined by the virulence of the influenza virus and the state of the patient's immune system. Clinics. The incubation period lasts from 12 to 48 hours. Typical flu begins acutely. Patients complain of chill, fever, weakness, pain in muscles, joints, eyes, sweating, severe headache, possibly dryness, discomfort in the throat. Catarrhal syndrome from the mucous membranes of the upper respiratory tract can develop both a few hours after the onset of the disease, and on the second day, which manifests itself in the form of a runny nose, cough. On examination, such complaints as hyperemia of the face, vascular injection of the sclera, increased sweating, body temperature, decrease of blood pressure are revealed. Rhinitis, pharyngitis, tracheitis, laryngitis are possible. The disorder of the oral mucosa is nonspecific and depends on the type of virus, the reactivity of the body, and local immunity. Thus, and at initial stage of influenza hyperemia of mucosa of the soft palate palatine arches, pharynx, sometimes buccal mucosa and gums is observed. At the same time, military granular eruptions of red color are present, which are formed as a result of hyperplasia of the epithelium of the excretory ducts of the salivary glands. After a few days, lesions of the mucous membrane are present. Catarrhal stomatitis develops. A sharp decrease in immunity, characteristic of acute viral infection, poor hygiene of the teeth can lead to activation of the resident microflora of the oral cavity. Layering of a secondary infection can lead to the development of acute herpetic, ulcerative-necrotic, aphthous, candidal stomatitis. As a complication, neuritis of the trigeminal and facial nerves sometimes occurs. Recovery in such cases is delayed for 2-3 weeks. Peripheral blood is characterized by leukopenia, eosinopenia, neutropenia, relative monocytosis, lymphocytosis, ESR can be increased. An immunofluorescence diagnosis of the influenza pathogen is found in the mucus of the nose or pharynx. Confirmation of the diagnosis is also possible by serological diagnostic methods ( serum of patients should be taken with an interval of 5-7 days). Treatment. Patients should be isolated and follow bed regimen. Careful oral hygiene is required. Local interventions in the presence of changes in the mucous membrane of the

oral cavity are determined by the type of stomatitis that occurs (catarrhal, herpetic, aphthous, ulcerative necrotic, candidal, etc.) and are aimed at preventing the attachment of a secondary infection (the use of local antiseptic agents). Prevention. For specific influenza prophylaxis, influenza vaccination is indicated, which is carried out before the epidemic period.

Rubeola (measles) is an infection caused by a virus that grows in the cells lining the throat and lungs. It's a very contagious disease that spreads through the air whenever someone who is infected coughs or sneezes.

People who catch the measles develop symptoms such as a fever, cough, and runny nose. A telltale rash is the hallmark of the disease. If measles isn't treated, it can lead to complications such as ear infection, pneumonia, and encephalitis (inflammation of the brain).

Koplik's spots



Measles complications the measles develop complications such as pneumonia, ear infections, diarrhea, and encephalitis treatment for measles. Sometimes getting the measles, mumps, and rubella (MMR) vaccine within the first three days after being exposed to the virus can prevent the disease.

The best advice for people who are already sick is to rest and give the body time to recover. Stay comfortable by drinking plenty of fluids and taking acetaminophen (Tylenol) for fever.

## TOPIC 12.

## INFECTIOUS MONONUCLEOSIS. MURRAIN. ETIOLOGY, PATHOGENESIS, CLINICAL MANIFESTATIONS OF THE ORAL MUCOSA, DIAGNOSIS, TREATMENT AND PREVENTION.

Murrain is an acute viral disease transmitted to humans from infected animal and is characterized by fever, general intoxication and bubble-aphthous lesions of the mucous membranes, skin peeling. Etiology, pathogenesis. The causative agent of the disease is Picornavirus, which is well preserved in dried, frozen, slightly sensitive to alcohol, ether, inactivated by heating. The source and the reservoir of infection are infected patients, patients who have contact with domestic and wild animals (cows, goats, sheep, pigs, deer, etc). Saliva, milk, urine, stool are sources for transmission, and it can be stored for up to 2-3 months. A person is infected when he /she has eaten raw dairy products, meat that has undercooked. Boiling, pasteurizing milk destroys the virus. Infection can occur from direct contact with a sick animal when caring for it. Diseases from person to person are not transmitted. Children are most susceptible to the virus. The sources of infection are oral mucosa, nose, eyes, and damaged skin. Clinisc. The incubation period lasts from 2 to 12 days. The formation of the primary effect at the site of the deepening of the virus occurs without temperature rise. After the virus enters the blood, the body temperature rises sharply to 39C, chills, weakness, pain in the muscles, joints, in the buccal area, loss of appetite, and vomiting are observed. After 1-2 days, patients complain of dryness and burning in the mouth, redness of the eyes, painful urination. The history taking of the disease is important to assess the epidemiological situation (contact with sick animals, the use of raw milk). New vesicles can be present as secondary signs of this disease. They appear in the oral cavity, around the mouth, in the folds, fingers, nails, genitalia, and conjunctiva. So, the oral mucosa is edematous, hyperemic, and is covered with a whitish-yellow coating, salivation increases, and bad breath occurs. Numerous vesicles with a diameter of 2-4 mm, filled with milky contents, appear on the entire mucosa, but there are more on the lips, tongue, palate. They are relatively thickwalled and therefore remain in the oral cavity for 1-2 days, and then burst with the formation of painful superficial erosions surrounded by a bright red rim. On the lips such erosions are covered with crusts. But after falling down body temperature the patient's condition does not improve. Increased salivation (to 4-5 per day), painful lesions in the mouth, edema, difficult swallowing are observed. Regional lymph nodes increase, and become sensitive. Rash of vesicles on the skin is accompanied by burning, itching in areas of the hands and feet. Erosions on the mucous membranes are epithelized after 2-5 days without leaving scarring, after which the general condition of the patient is normalized and a period of recovery begins, lasting 10-15 days. Relapses of rashes of lesion elements on the skin and mucous membranes are possible, which stop recovery for several months. For laboratory confirmation of the diagnosis, a biological test is developed and performed, serological reactions are infected in laboratory animals. Treatment. Compulsory isolation and hospitalization of the patient is required and lasts for 14 days. There is no specific general treatment. Prescribe vitamins, painkillers. Local interventions are similar to the treatment of acute herpetic stomatitis. Prevention. Vaccination, disinfection measures are necessary and required. Safety measures when caring for sick animals are required. Do not use dairy and meat products from sick or suspicious animals. To prevent infection through food, milk should be boiled for 5 minutes and the meat should be thoroughly cooked. Do not work on farms, where there can be cases of murrain.

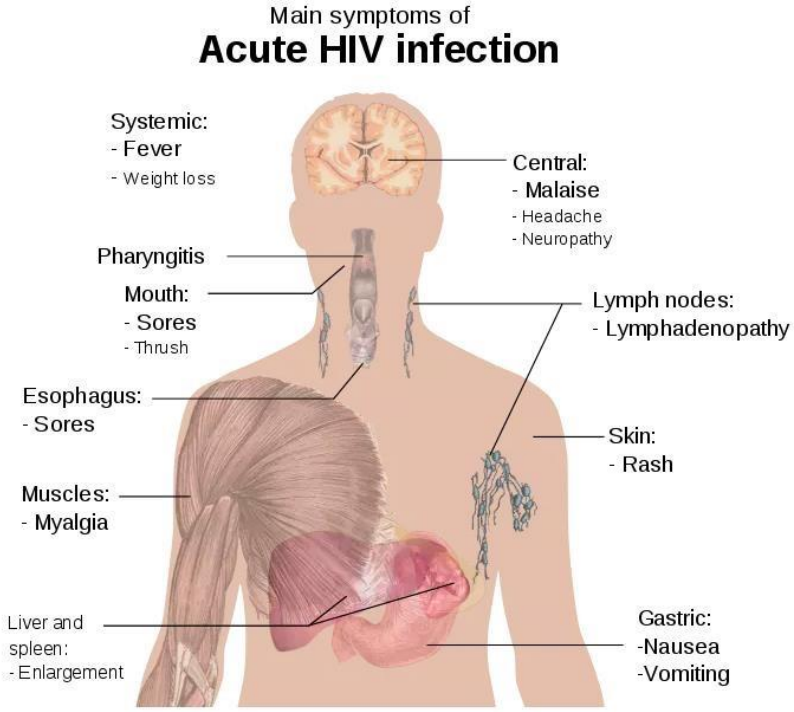
INFECTIOUS MONONUCLEOSIS (mononucleosis infectiosa, FilatovPfeiffer disease) is an acute viral disease characterized by damage to the reticuloendothelial system with fever, tonsillitis, polyadenitis, hepatosplenomegaly, and lymphocyte blast transformation. Etiology, pathogenesis. Diseases are not very contagious due to the high percentage of immune individuals

(antibodies are detected in 80% of adults), the presence of asymptomatic forms. The source of infection is a sick person. Infection occurs by airborne droplets, and transfusion transmission through the blood is also possible. Sick children, young people are susceptible to it. The causative agent of infectious mononucleosis is the human herpes virus type 4 (Epstein-Barr virus), which has a tropism for B-lymphocytes. It can persist for a long time in the host cells as a latent infection. The source infection is the mucous membrane of the upper respiratory tract. The causative agent particularly damages the lymphoid and reticular tissue, which leads to generalized lymphadenopathy, enlargement of the liver, spleen, and changes in the blood. In pathogenesis, an important role is assigned to the stratification of a secondary infection, especially in patients with necrotic changes in the pharynx. Clinics. The incubation period lasts from 4 to 15 days, but can be up to 50 days. The disease usually begins acutely. Body temperature rises rapidly to 39- 40C. The temperature curve is wrong type, sometimes with a tendency to wave, the duration of the fever lasts 1-3 weeks, it comes gradually. From the first days, weakness, headache, myalgia, arthralgia are present. The patient's face is puffy, nasal breathing is difficult, nosebleeds are possible. Firstly, increased cervical lymph nodes, submandibular, sometimes - axillary, inguinal, cubital. The nodes sometimes reach the size of a “chicken egg”, mobile, slightly painful, densely elastic, not gathered together and with surrounding tissues. Around the cervical nodes there may be edema, however, the skin remains without signs of inflammation. 3-5 hours a quarter of patients revealed rash, this rash appears on the skin of the trunk, limbs. Elements of damage after 1-3 days disappear without any sign. Along with this, the liver and spleen significantly increase and become dense. The pharynx of patients is sharply hyperemic; tonsil hyperplasia appears. Tonsillitis develops at different stages of infectious mononucleosis and can be catarrhal, lacunar, ulcerative necrotic with the formation of fibrinous films resembling diphtheria. On 3-4 days of illness, petechiae appear on the border of the hard and soft palate, the development of catarrhal, herpetic or ulcerative necrotic stomatitis is possible. Tongue is coated with gray-white coating, expressed hyperplasia linguistic the tonsils, fungal-shaped papillae. In peripheral blood, leukocytosis is detected up to 15-20x109 /l (less commonly leukopenia), neutropenia, an increase in the percentage of lymphocytes, monocytes, eosinophils. The appearance of atypical mononuclear cells up to 15- 30% of all leukocytes (cells with a nucleus of a large lymphocyte and a broad basophilic cytoplasm with a violet hue with pronounced perinuclear enlightenment), which can exist for several months, is characteristic. The content of erythrocytes and hemoglobin does not change significantly, ESR is 20-30 mm/h. There are virus-specific serological studies that are performed in cases difficult for diagnosis. They constitute modifications of the heteroagglutination reaction. A specific confirmation of the diagnosis of infectious mononucleosis is the identification of an increase in antibody titer of 4 times or more for Epstein-Barr virus. Treatment. Patients do not require special drug therapy. Generally, they are limited of vitamins use, symptomatic agents of general and local effects (antiseptics, anti-inflammatory, painkillers, keratoplastic drugs). Chloramphenicol, sulfonamides are contraindicated, because they inhibit hematopoiesis. Antibiotics are used in cases of secondary bacterial infection. In severe forms of infectious mononucleosis, corticosteroid drugs are used. Prevention. Persons who have undergone infectious mononucleosis for a long time cannot be donors. Preventive measures in the focus of infection are not carried out.

## TOPIC 13.

## AIDS. ETIOLOGY, PATHOGENESIS. MANIFESTATIONS ON THE ORAL MUCOSA, DIAGNOSIS. TREATMENT AND PREVENTION. DENTIST'S TACTICS.

AIDS. Acquired immune deficiency syndrome (AIDS) is an infectious disease caused by the human immunodeficiency virus (HIV). There are two variants of the HI V virus, HIV-1 and HIV-2, both of which ultimately cause AIDS.




Main symptoms of acute HIV infection

Diagnosis. HIV/AIDS is diagnosed via laboratory testing and then staged based on the pr esence of certain signs or symptoms. HIV screening is recommended by the United States Preventive Services Tas k Force for all people 15 years to 65 years of age including all pregnant w ome n. Additionally, testing is recommended for those at high risk, which includes any one diagnosed with a sexually transmitted illness.

In many areas of the world, a third of HIV carriers only discover they are infect ed at an advanced stage of the disease when AIDS or severe immunodeficien cy has become apparent Treatment

There is currently no cure or effective HIV vaccine. Treatment consists of highly active antiretroviral therapy (HAART) which slows progression of the disease.

As of 2010 more than 6.6 million people were taking them in low and middle income countries.[144] Treatment also incl udes preventive and active treatment of opportunistic infections.

## TOPIC 14.

## DIPHTHERIA. SCARLET FEVER. CHICKEN POX. ETIOLOGY, PATHOGENESIS, CLINICAL MANIFESTATIONS OF THE ORAL MUCOSA, DIAGNOSIS, TREATMENT AND PREVENTION.

Diphtheria is an acute infectious disease that is transmitted by airborne droplets. The causative agent is Leffler's diphtheria bacillus, the pathogenic properties of which are determined by its exotoxin. Clinically the incubation period is 2-10 days. Then there is a sore throat, t38-390 C, general weakness, heart pain, lack of appetite. From the first hours of the disease, hyperemia and swelling of the mucous membrane of the tonsils (diphtheria angina) develops. Then massive fibrinous films of white or grayish-white color are formed, which extend to the mucous membrane of the nasal part of the pharynx, the hard palate and can spread to the gums, mucous membrane of the cheeks, tongue. The film coating is tightly soldered to the underlying tissues, has a sweetish smell and is very difficult to remove, exposing the bleeding surface. The occurrence of films is associated with a fibrinous form of inflammation and is a local reaction to the deepening of Leffler's bacillus and its toxins. Diagnosis of the disease is based on data from a bacteriological study, conducting a passive hemagglutination reaction. Differential diagnosis - manifestations of scarlet fever, infectious mononucleosis, Simanovsky-Vincent sore throat, acute herpetic stomatitis, erythema multiforme exudative, acute candidiasis, leukemia. Treatment of patients is carried out in a hospital! infectious diseases hospital, and consists in the introduction of diphtheria antitoxin, anti-inflammatory drugs, vitamins, heart drugs. Locally use antiseptics, antibiotics, enzymes, painkillers and keratoplastic agents. For the prevention of diphtheria, toxoid vaccinations are important.

Treatment

Diphtheria is a serious condition, so your doctor will want to treat you quickly and aggressively.

The first step of treatment is an antitoxin injection. This is used to counteract the toxin produced by the bacteria. Make sure to tell your doctor if you suspect you might be allergic to the antitoxin. They may be able to give you small doses of the antitoxin and gradually build up to higher amounts. Your doctor will also prescribe antibiotics, such as erythromycin or penicillin, to help clear up the infection.

During treatment, your doctor may have you stay in the hospital so you can avoid passing your infection on to others. They may also prescribe antibiotics for those close to you.

Scarlet fever

Scarlet fever is a disease caused by an exotoxin released by Streptococcus

pyogenes.

It is characterized by sore throat, fever, a 'strawberry tongue', fine sandpaper rash over the upper body that may spread to cover almost the and uvula (Forchheimer spots).

Characteristic rash, which: is fine, red, and rough-textured; it blanches upon pressure appears 12–48 hours after the fever generally starts on the chest, axilla (armpits), and behind the ears is worse in the skin folds Pastia lines (where the rash becomes confluent in the arm pits and groins) appear and persist after the rash is gone.

The rash begins to fade three to four days after onset and desquamation (peeling) begins. "This phase begins with flakes peeling from the face. Peeling from the palms and around the fingers occurs about a week later." Peeling also occurs in axilla, groin, and tips of the fingers and toes.

Treatment of scarlatina is etiotropical therapy. Prescription of antibiotics. Palliative mouthrinses may be helpful. Early diagnosis and treatment are important to prevent complications, which include local abscess formation, rheumatic fever, arthritis, and glomerulonephritis.

Chicken pox.

The most distinctive sign of chickenpox infection is an itchy rash of red spots and blisters. It takes about 1 or 2 days for a chickenpox red spot (macule) to go through all of its stages, including blistering, bursting, drying, and crusting over.

## TOPIC 15.

## TUBERCULOSIS. ETIOLOGY, PATHOGENESIS, CLINICAL MANIFESTATIONS OF THE ORAL MUCOSA, DIAGNOSIS. TREATMENT AND PREVENTION. DENTIST'S TACTICS.

Tuberculosis is a chronic infectious disease caused by mycobacterium tuberculosis (Koch's bacillus). It enters the mucous membrane of the mouth by the hematogenous, lymphogenous or exogenous route. In the oral mucosa, tuberculosis occurs in the form of a secondary lesion and manifests itself in the form of: lupus erythematosus tuberculosis, miliary ulcerative tuberculosis, colic tuberculosis (scrofuloderma). Tuberculosis lupus. The main element of the lesion is lupoma, a specific tuberculous tubercle (tuberculum), red or yellow-red, soft consistency with a diameter of 1-3 mm. Lupomas are located in groups, fresh ones are formed on the periphery, and those in the center are prone to decay, after which ulcers with soft, uneven, saped edges, swollen and less painful form. The bottom of the ulcer is covered with yellow-red raspberry-like growths that bleed easily. Stages of the process: infiltrative, hilly, ulcerative and cicatricial. Localization of the elements of the lesion: red border of the upper lip, gums and alveolar process of the upper jaw in the region of the front teeth and fangs. Sometimes the process moves to a hard and soft palate. Regional lymph nodes are enlarged, dense, bundled. Diagnosis: a symptom of "apple jelly" during dioscopy and a symptom of a probe failing in a lupoma (Pospelov phenomenon). The reaction of Pirke is positive. Histological examination reveals epithelioid cells, giant Pirogov-Lanhgans cells and peripheral lymphocytes. Differential diagnosis: manifestations of tertiary syphilis (tubercle syphilis), leprosy, lupus erythematosus. Miliary - ulcerative tuberculosis. It occurs in patients with severe forms of pulmonary tuberculosis or larynx. Mycobacterium tuberculosis with the sputum of the patient settles in the places of the oral mucosa, which is prone to injuries (back of the tongue, mucous membrane of the cheeks along the line of closure of the teeth, soft palate, clear). The microflora multiplies, and typical tuberculous tubercles arise, which decay in the center and form shallow ulcers, which are creeping in nature, with uneven soft undercut edges. The bottom and edges of the ulcer are granular (due to tubercles), covered with a yellow-gray coating. Small abscesses (Trill grains) are determined. Inflammation around the ulcer is weak. Lymph nodes are enlarged, tight - elastic, painful. Diagnosis: the general condition of the patients is important (weight loss, excessive sweating, shortness of breath, fever) elevated ESR in the blood, leukocytosis, lymphocytosis. In scrapings from ulcers, Pirogov-Lanhgans cells are found, with bacteriological examination - Koch bacilli. Differential diagnosis: performed with a gummy ulcer in syphilis, Vincent’s necrotic stomatitis, radio mucositis, a traumatic chronic ulcer, trophic, cancer ulcer and manifestations of secondary syphilis. 5 Colquatic tuberculosis (scrofuloderma) a form of secondary tuberculosis. The main element of the lesion is the node that forms in the deep layers of the mucous membrane. 3 time, the nodes disintegrate and ulcers of irregular shape, soft consistency arise, with eaten sap edges and sluggish granulations at the bottom. Ulcers are slightly painful; uneven shaggy scars form during healing. Differential diagnosis: gummous, cancerous, trophic ulcers, Seton's stomatitis, actinomycosis. Tactics of the dentist: if the dentist diagnosed the patient with tuberculosis, he should send him for a consultation with a TB doctor. When confirming the diagnosis, treatment is carried out in a tuberculosis dispensary

Treatment medications for active TB disease include:

- isoniazid
- ethambutol (Myambutol)
- pyrazinamide
- rifampin (Rifadin, Rimactane)
- rifapentine (Priftin)

## TOPIC 16.

## SYPHILIS. GONORRHEA. ETIOLOGY, PATHOGENESIS, CLINICAL MANIFESTATIONS OF THE ORAL MUCOSA, DIAGNOSIS. TREATMENT AND PREVENTION. DENTIST'S TACTICS.

Syphilis is a chronic infectious disease caused by a pale spirochete (spirochete palida). In the oral cavity, it manifests itself in all stages of the disease: primary, secondary and tertiary syphilis. Primary syphilis is a hard chancre that can be localized in various parts of the oral cavity, mainly on the lips, tongue, and corners of the mouth. The incubation period lasts 14-20 days. Clinically. First, erosion of a bright red color occurs, then a defect in the form of an ulcer. An infiltrate forms around the lesion. The edges of the ulcer are raised, roll-shaped, in connection with which the solid chancre rises above the level of the mucous membrane. On palpation, a painless cartilage seal is felt. Regional lymph nodes are dense, mobile, painless. Diagnostics. When bacteriological examination of the lesion is found pale spirochete. Differential diagnosis: tuberculous ulcer, trophic, decubital, cancer ulcer. Secondary syphilis in the oral cavity manifests itself in the form of individual roseola, or erythema (roseolous syphilis), or papules (papular syphilis) and less often pustules - pustular syphilis. Diagnosis of secondary syphilis is confirmed by the presence of pale treponema in the lesions and positive serological reactions. Differential diagnosis: lichen planus, leukoplakia, allergic stomatitis. Tertiary syphilis manifests itself in the oral cavity: in the form of gum, tubercular syphilis, sclerosed glossitis. Gumma is a node that clearly protrudes above the level of the oral mucosa the size of a bean, red in color with a dense consistency. Gradually, the color gains a bluish tint, an infiltrate is created, which turns into bone necrosis of the hard palate, a sequestration develops, which exfoliates and a message forms between the oral cavity and the nose. If the gumma on the tongue, then it is laid in the linguistic muscles. When it decays, an ulcer forms, which has inclined and dense edges. The ulcers are deep, painless, and crater-like, with a dirty gray bottom. With the reverse development, they heal with the formation of deep retracted scars. 6 Differential diagnosis: with cancer, tuberculous ulcers and decubital. Sclerosing glossitis from the folded tongue. Primary and secondary syphilis are easy to treat with a penicillin injection. Penicillin is one of the most widely used antibiotics and is usually effective in treating syphilis. People who are allergic to penicillin will likely be treated with a different antibiotic, such as: doxycycline azithromycin ceftriaxone.

Gonorrhea is a sexually transmitted infection (STI). It’s caused by the bacterium Neisseria gonorrhoeae Symptoms usually occur within 2 to 14 days after exposure. However, some people who acquire gonorrhea never develop noticeable symptoms.

Gonorrhea is an acute infectious disease of the oral mucosa caused by gonococcus. The incubation period is from 1 day to 1 month. After 3-4 days, gonococci that enter the oral mucosa reach the subepithelial layer of connective tissue through the intercellular spaces and cause an inflammatory reaction with the formation of purulent exudate, considered as migration of neutrophils and plasmocytes to the pathogen invasion site. Complaints of patients are absent. The mucous membrane of the lips, gums, lateral and lower surface of the tongue and the bottom of the oral cavity, pharynx, tonsils, larynx is brightly hyperemic and covered with a dirty - gray, sometimes greenish purulent coating with an unpleasant odor. Often, unilateral arthritis of the jaw-temporal joint develops, characterized by significant pain, then acute inflammation of the joint develops, swelling appears, the skin in the joint area turns red; it becomes tense and sharply painful. Diagnosis is confirmed by the presence of gonococcus with exudate microscopy. 7 Differential diagnosis is carried out with diphtheria, drug allergic stomatitis, erythema multiforme exudative, ulcerative stomatitis, fungal stomatitis. General and local treatment consists in taking antibiotics

(ceftriaxone, cefazolin, cefabid, ciprinol) for 1-2 weeks. Topically applied enzymes, antiseptic irrigation. The main therapy is carried out by a dermatovenerologist

**TOPIC 17.**

**MODULE**

**TOPIC 18.**

## CHANGES IN THE MUCOUS MEMBRANE OF THE ORAL CAVITY IN DISEASES OF THE DIGESTIVE CANAL (PEPTIC ULCER, GASTRITIS, ENTERITIS, COLITIS, CHRONIC HEPATITIS).

Changes in the oral mucosa at diseases of gastrointestinal tract Changes in the tongue. They are the most typical at diseases of gastroin- testinal tract.

Among these signs the furred tongue is most frequently revealed. The disturbance of the process of cornification and destruction of epithelial cells in the tongue papillae, as a result of the neurotrophic disorders, play an important role in the formation of fur on the tongue. The nature of food, the composition of microflora, the oral hygiene influence the accumulation of fur. If the epithe- lium detachment is not observed, the keratinized cells remain, dead leukocytes and microorganisms of saliva are joined and form fur. In this case the favorable conditions for multiplication of microorganisms are created, in particular, a sig- nificant amount of fungi is formed. All of this is the basis of fur. Fur is revealed with gastritis, stomach ulcer and duodenal ulcer, new formations in the stomach and other illnesses. Edema of the tongue is the second sign of gastrointestinal diseases. It does not cause subjective sensations in patient. Edema of the tongue is observed in case of chronic bowel diseases, which is explained by the disturbance of the sucking ability in the gut and gut barrier function. The changes in the different groups of the papillae of the tongue are observed at diseases of gastrointestinal tract. Depending on their state hyper- and hypoplastic glossitis are distinguished.

Hyperplastic glossitis is observed in patients in case of gastritis with increased acidity. It is accompanied by the hypertrophy of papillae, presence of fur, a slight increase in size of the tongue as a result of edema.

Hypoplastic glossitis is characterized by the atrophy of papillae, sometimes sharply expressed, in consequence of which it become varnished, with bright spots and strips. The atrophy of the tongue papillae causes the sensation of burning, tin- gling, pain when eating. Such changes in the tongue papillae are observed in case of gastritis with lowered secretion, ulcer, gastroenteritis, biliary tract disease.

## TOPIC 19.

## CHANGES IN THE MUCOUS MEMBRANE OF THE ORAL CAVITY IN DISEASES OF THE CARDIOVASCULAR SYSTEM (CIRCULATORY INSUFFICIENCY, HYPERTENSION).

Trophic ulcer. Bladder-vascular syndrome. Lesions of the oral mucosa at cardiovascular pathology Changes of oral mucosa at cardiovascular diseases depend on the degree of insufficiency of blood circulation, state of vascular wall, etc. Cyanosis of oral mucosa, lips and the tongue of bright red or crimson color are characteristic for the acute period of myocardial infarction.

The appearance of trophic changes of oral mucosa, up to the formation of ulcers, is observed predominantly in patients with the decompensated defects of heart and the disturbance of blood circulation of III, sometimes II degree.

Trophic ulcers are localized predominantly in the rear of the mouth, on the cheek mucosa, alveolar branch, on the tongue, on the mucous a pearshaped area, etc. Ulcers have different sizes (2-5 mm and more). They are covered with pale gray fur with the fetid smell; it is sharply painful when touching and eating. The inflammatory reaction in the surrounding tissues is absent. The treatment of such changes of oral mucosa provides for the liquidation of the insufficiency of blood circulation in combination with the local symptomatic therapy.

## TOPIC 20.

## CHANGES IN THE MUCOUS MEMBRANE OF THE ORAL CAVITY IN DISEASES ENDOCRINE SYSTEM (ACROMEGALY, ITSENKO-CUSHING'S DISEASE, DIABETES).

Xerostomia.

Changes in the oral mucosa at endocrine diseases Diabetes mellitus. The most characteristic changes in the oral cavity are xero- stomia, catarrhal stomatitis and glossitis, fungal stomatitis, mycotic perleche, paresthesias of oral mucosa, trophic disorders, Lichen ruber planus. Dentist conducts the treatment of a patient together with the endocrinologist, symptomatic treatment is prescribed with the expressed changes in the oral cavity taking into account their manifestation.

Myxedema is developed with the insufficiency of the function of the thyroid gland. The face of patient takes the unique form: lips and nose are thickened, upper The eyelids are sharply edematic, and facial expression is indifferent. In patients ane- mia, edema and dryness of oral mucosa are observed. Myxedema is accompanied by a marked increase of the tongue, which is sometimes not placed in the oral cav- ity, an increase of the lips, gums. Dentist conducts the sanitation of the oral cavity and symptomatic treatment if necessary.

Pregnancy gingivitis is an inflammation of gums, which first appears dur- ing pregnancy or is exacerbated by pregnancy. The development of the disease is connected with restoring of hormonal balance during this period. In the first half of pregnancy the catarrhal gingivitis is noted. In the second half - the course of dis- ease is heavy, with the development of proliferating process in the gums. In the initial stage of gingivitis the gingival edge becomes clear red, swells, bleeds easily. Gradually the affected gum becomes dark red, cyanotic, increases and with the pres- ence of local stimuli the hypertrophic gingivitis develops. Hypertrophic gingivitis in pregnancy tends to polypous growth of separate papillae. Sometimes false epulis develop. The hypertrophied gum covers the entire dental crown, bleeds easily. Treatment. The local treatment of pregnancy gingivitis is conducted through the principles of the treatment of catarrhal or hypertrophic gingivitis.

Itsenko-Cushing disease. The oral mucosa in such patients is edematic, the imprints of teeth on the tongue and the cheeks are observed. Appearing trophic disorders lead to the appearance of erosions and ulcers which are characterized by a prolonged course. Candidiasis is frequently observed.

## TOPIC 21.

## CHANGES IN THE MUCOUS MEMBRANE OF THE ORAL CAVITY IN DISEASES OF THE BLOOD AND BLOOD-FORMING ORGANS. LEUKEMIA. AGRANULOCYTOSIS. DENTIST'S TACTICS.

Leucosis is the malignant disease of the hemopoietic organs, which appears as a result of progressive cellular hyperplasia in the hemopoietic organs, when the processes of cellular division (proliferation) prevail above the processes of ripening (differentiation). Pathomorphological substratum of disease is leukemia blast cells which correspond to primordial elements of one of the hematopoietic lineage.

The clinical picture of acute leucosis determines 4 leading syndromes: hem- orrhagic, hyperplastic, anaemic and intoxicating. Hemorrhagic syndrome is manifested in the form of petechiae, ecchymosis, hematomas on the skin and mucous membrane, or the profuse bleedings. In the oral cavity the most characteristic are: acute bleeding of gums, pres- ence of hemorrhages on mucous membrane of checks along the line of the join- ing of teeth, in the tongue, the palate. Severe hemorrhages and hematomas some- times are revealed.

Hyperplastic processes are manifested in the increase of the lymph nodes, liver, spleen, tonsils. Frequently hyperplasia is combined with ul- ceronecrotic changes in the gums. Furthermore, necroses are revealed also in the tonsils, the pear-shaped area and other parts of oral mucosa. Its tendency toward the propagation in the adjacent sections is the special feature of necrotic process in case of sharp leucosis, in consequence of which the unlimited ulcers of irregular outlines, covered with gray necrotic fur appear. Reactive changes around the ulcer are absent or are weakly expressed. The development of ulceronecrotic processes in the oral cavity is connected with sharp reduction in the resistibility of tissues caused by reduction in the phagocytic activity of leukocytes and immune properties of blood serum. Patients with acute leucosis complain about pain in the intact teeth and the jaws (together with pain in other bones), which is explained by the direct lesion of the bones in case of the leukemic process. Treatment is conducted in the hematology hospitals. The treatment of leukosis stomatitis is symptomatic. The extraction of teeth is contraindicated.

The chronic leucosis appear more rarely than acute, development is more fa- vorable; course is prolonged. Chronic myeloleukemia passes two stages: benign (lasts several years) and malignant (terminal), which lasts for 3 -6 months. The basic sign of chronic my- eloleukemia in the oral cavity are hemorrhagic manifestations, but with consider- ably smaller intensity, than in case of acute leucosis. Bleeding of gums appears not spontaneously, but only with the traumatization, the removal of teeth. In the period of excerbation the ulceronecrotic lesions of oral mucosa are observed. Chronic lymphoid leukosis is characterized by slow beginning and prolonged latent course. In the initial stage of disease an increase in groups of lymph nodes is observed. At the developed stage the generalized increase of lymph nodes is observed, the pallor of the skin and the mucous membranes, leukemic infiltrations of gums. the tongue, and hyperplasia of interdental papillae appear. Sometimes the growth of gingival edge reaches the level of the joining of teeth. Treatment is conducted in the hematology departments. Local treatment consists in care of the oral cavity, full-fledged sanitation, symptomatic therapy.

Agranulocytosis is a syndrome, which is characterized by the significant de- crease in the number or absence of neutrophilic granulocytes in the peripheral blood. Ulceronecrotic process in the lips, the gums, the tongue, mucous membrane of cheeks and other sections develop. Ulceronecrotic process can spread into the gullet. The absence of the inflammatory reaction of tissues around the centre of necrosis is im- portant for diagnostics. Frequently necrotic process is combined with candidiasis. Treatment is conducted in the hematology departments. Local treatment

is symptomatic; it includes the antiseptic treatment of oral cavity, anesthetization, removal of necrotic tissues, and prescription of the preparations, which stimulate regeneration.

## TOPIC 22.

## CHANGES IN THE MUCOUS MEMBRANE OF THE ORAL CAVITY IN DISEASES OF THE BLOOD AND BLOOD-FORMING ORGANS. ANEMIA. VASEZ'S DISEASE. WERLHOF'S DISEASE. DENTIST'S TACTICS.

Manifestations on oral mucosa with the diseases of the blood and hematopoietic organs Iron-deficiency (hypochromic) anemia (chlorosis).

Patients with early chlorosis complain about the disturbance of gustatory senses, sense of smell, reduction in appetite, nausea. The oral mucosa is without the noticeable disturbances, except for color change it becomes pale. All symptoms are more expressed with late chlorosis. Patients complain about painful sensations in the tongue and oral mucosa when eating sour or spicy food, dryness in the oral cavity, paresthesia (burn- ing, tingling, prickling and bursting the tongue open), and the presence of angular heilitis.

The clinical picture of hypochromic anemia is multiple lesions of carious teeth, their increased abrasion, and loss of the natural luster of enamel. Mucosa membrane of lips, cheeks and gums is pale, edematic. The tongue is edematic, papillae are atrophied, espe- cially in its front part. It becomes clear red and smooth, as if polished, deep folds appear. Sometimes hemorrhages and cracks in the angles of the mouth are observed.

Treatment. The symptomatic therapy of manifestations on mucous membrane is prescribed. Hypoplastic anemia appears under the action of the exogenous physical (ir- radiation) and chemical factors, and drugs as well as endogenous aplasia of bone marrow. On the background of particularly pale oral mucosa different heavy hem- orrhages appear. Interdental papillae are edematic, cyanotic, sometimes bleeding, deep parodontal pockets are revealed. On oral mucosa, besides petechiae, also ero- sions, ulcers, necrotic sections are observed. Treatment. Dentist conducts the symptomatic treatment of the manifestations of hypoplastic anemia.

B12 - folate deficient anemia (malignant anemia, pernicious anemia, Addison – Birmera disease) is characterized by the disturbance of erythropoiesis. The clinical picture of disease consists of the triad: the dysfunction of the gastrointestinal tract, hemopoietic and nervous systems. One of the early symptoms of the disease is the pallor of the skin and oral mucosa with a yellowish tint. Sometimes on oral mucosa petechial hemorrhages are observed. The most characteristic lesion of oral mucosa in case of malignant anemia is hunter's glossitis. The back of the tongue in this case takes the form of the smooth, bright, polished surface as a result of the atrophy of mushroom-shaped filamentary papillae, thinning of epithelium and atrophy of muscles.

Painful, sharply limited strips and spots of clear red color of inflammatory nature appear at the back and the tip of the tongue. Treatment is carried out in the hematology clinics. Locally symptomatic treat- ments, sanitation of oral cavity are carried out.

Erythremiya (polycythemia, the Vaquez' disease) Erythremiya appears at the age of 40-60 years, predominantly in men. Disease begins unnoticeably, and develops slowly. Increased fatigue, bleeding of gums, hemorrhages from the nose are noted. Characteristic symptom for patients is dark-cherry polycythemia of oral mucosa. Lips, the tongue, buccal mucosa are clear red because of the increased content of reduced hemoglobin in the capillaries. Mucous membrane in the region of alveolar branches is friable, with the cyanotic tint; when pressing bleeds easily, interdental papillae are hyperemized, and of dark- cherry colour. Expressive color boundary - cyanosis of soft palate and pale color of hard palate (Cooperman's symptom) is characteristic. The itching of the skin and paresthesia of oral mucosa due to the increased filling of vessels and the irritation of the interoceptors of capillaries by the blood is possible. Treatment is conducted by hematologist,

making periodic bloodlettings and us- ing the cytostatic therapy with radioactive phosphorus or mielosan.

Thrombocytopenic purpura (Verlgof disease) The basic clinical symptom of disease is hemorrhages from the skin and oral mu- cosa, as well as nose and gums, that appear spontaneously or under the effect of the insignificant injury. In general in case of thrombocytopenic purpura the oral mucosa is pale, edematic, atrophied; the thinning of epithelium, erosions or ulcers form. Treatment is conducted in the hematology departments. Changes in the oral mucosa at hypo- and avitaminoses

## TOPIC 23.

## CHANGES OF A MUCOUS MEMBRANE OF AN ORAL CAVITY AT HYPO-AND AVITAMINOSIS A, C. TACTICS OF THE DENTIST.

Hypovitaminosis A

Vitamin A (retinol) is of great importance in the processes of growth and devel- opment of organism; it regulates the processes of ripening and keratinization of the epithelium, increases resistance of oral mucosa to the action on it of different trau- matic and irritating factors, ensures the normal functioning of the organ of vision, favors the normal functioning of salivary and sweat glands. The clinical manifesta- tions of hypovitaminosis are caused by reduction in the barrier properties of the skin and oral mucosa, the disruption of the normal differentiation of epithelial tissues. At hypovitaminosis A oral mucosa is pale, turbid, dry, loses its characteristic luster. On mucous membrane of cheeks, the hard and soft palate the whitish stratifications, which resemble the mild form of leukoplakia appear. Keratinization of epithelium of the excretory ducts of salivary glands occurs. This leads to a decrease in secretion of saliva - hyposalivation. Treatment - products rich in vitamin A (butter, milk, egg yolk, sour cream, liver), or carotene, which is converted in the organism into the vitamin A (carrot, apricots, peaches, black currant). Fish oil (3 tablespoons per day for adult person, 3 teaspoons - for the child) is prescribed.

Hypovitaminosis C

The insufficiency of vitamin C is always manifested by changes in the oral cav- ity. Scorbutic stomatitis is one of the earliest and frequent symptoms of the disease. Three stages of scorbutic stomatitis are distinguished:

scorbutic stomatopatia - the initial stage;

scorbutic reparative stomatitis - the developed stage; 3. scorbutic ulcerous stomatitis - the complicated stage.

Initial stage is characterized by expressed gingivitis on the background of pale anemic oral mucosa: gum becomes infiltrated, gingival edge acquires dark red col- or, and bleeds easily. Sometimes single, scattered petechia in gum and oral mucosa is observed. Simultaneously petechiae can be revealed on the extremities, in the shin region. Patients complain about general weakness, sleepiness, rheumatic pain in extremities, headache, and rapid fatigue. Initial stage is frequently accompanied by iron-deficiency anemia. The developed stage of the disease is characterized by the significant inflammation of gums. Gum is sharply edematic, enlarged, and friable; it covers a significant part of the dental crowns. Gingival edge acquires cyanotic tint, it bleeds considerably when pressing. On the edge of gingival papillae the blood clots are frequently accumulated. On oral mucosa the plural petechiae, ecchimozis are observed. The tongue is furred, swells sharply, and the teeth imprints on its edges are observed. Teeth are loosening. Petechiae, ecchymozis and massive hemorrhages are observed on the mucous membrane of cheeks, palate and in different parts of body.

The general state of patients deteriorates, they become adynamic, the face is pale with the earthen tint. The third, complicated stage of the disease appears with the connection of sec- ondary fusospirillary infection, in consequence of which scorbutic ulcerous stoma- titis develops, The granulations grown on the gingival edge reach the cutting edge or the occlusal surface of teeth; areas of necrosis and pitting appear on the gums. Gums bleed intensively; they are covered with necrotic fur, with the fetid smell. Ulcerous process extends to tongue, cheeks, lips, hard and soft palate. Teeth are loosening and fall out. The general state of patients is extremely heavy.

Without the treatment the disease can end lethally. Treatment, At the initial stage of the disease with correct treatment under the dispensary conditions scorbutic gingivitis disappears in 5-7 days. At the developed and complicated stages of the disease patients subject to hospitalization. Local treatment: the sanitation of oral cavity is indicated, careful oral hygiene, removal of dental caleulus and plaque, irrigation of the oral cavity by the dilute solu- tion of potassium permanganate, the solution of citral, 1 % solution of galaskörbin. Hypovitaminosis A Vitamin A (retinol) is of great importance in the processes of growth and devel- opment of organism; it regulates the processes of ripening and keratinization.

## TOPIC 24.

## CHANGES IN THE ORAL MUCOSA IN HYPO- AND AVITAMINOSIS GROUP B AND PP. DENTIST'S TACTICS.

Hypovitaminosis B1

A deficiency of vitamin B1 (thiamine) causes the disease, known as beri-beri. Changes in the oral cavity are not always sufficiently characteristic. The lack of thiamine is sometimes accompanied by the neuralgia of trigeminal nerve, increased by painful sensitivity of oral mucosa. Some patients complain about the pain. Fre- quently bubble lesions of oral mucosa develop, it is more similar to herpetic stoma- titis. Phials are localized on the hard palate and on the tongue. Treatment is specific: saturation of organism by thiamine. Local treatment is symptomatic

Hypovitaminosis B2

In the absence or deficiency of riboflavin (vitamin B,) in the food ariboflavino- sis develops. The disease is characterized by the inflammation of the lips (angular cheilitis), glossitis, and conjunctivitis. Angular cheilitis begins with the reddening in the angles of the mouth, and then the painful cracks appear, which resemble perleche. Lips become red, swol- len, cracks and erosions appear on their surface. Simultaneously glossitis devel- ops, which is accompanied by sharp painfulness. Filamentary papillae atrophy, thus clear red hypertrophied mushroom-shaped papillae appear across the back of the tongue. As a result of the atrophy of filamentary papillae the tongue becomes smooth, shiny; because of the decreased salivation it seems dry, with many grooves, increased in size. Besides glossitis, conjunctivitis appears, sometimes iritis, eyelids are inflamed, epiphora, burning in the eyes, visual acuity decrement are observed. Treatment consists in saturation of organism by vitamins of group B (B, B,, B2). Local treatment is symptomatic.

Hypovitaminosis PP

In the oral cavity the clinical manifestations of hypovitaminosis PP flow in several stages: the first stage is connected with the appearance of erythema; it is observed already in the prodromal period. Patients complain about the sensation of burning in the area of mucosa of lips, cheeks and especially the tongue. Oral mucosa becomes hyperemized, the tongue is swollen, and filamentary papillae are atrophied. The general state: weakness, headache, insomnia, loss of appetite appear. The pain in the mouth, like neuralgic, appears in stage II. Inflammatory process extends to entire oral mucosa. The tongue becomes clear red and edematic, with a raspberry-coloured tint, resembling the colour of cardinal's mantle - "car- dinal" tongue; the surface layers of the epithelium desquamate, thus the tongue be- comes bright, smooth, specular. Sometimes cracks appear on it - "chess tongue". At this stage the general state of the patient worsens, diarrhea, dermatitis, depres- sion, dystrophia appear. At the stage III the general state of patient is severe. In the oral cavity with the appearance of fuzospirill symbiosis the ulcerous stomatitis develops. Treatment is specific: saturation of the organism by nicotinic acid (daily dose of 150 mg), and at the 3rd stage dose increases to 300 mg, vitamins of group B: thia- mine (20-50 mg), riboflavin (10-20 mg), pyridoxine (50 mg) are prescribed.

## TOPIC 25.

## CHANGES IN THE MUCOUS MEMBRANE OF THE ORAL CAVITY IN DERMATOSES WITH AN AUTOIMMUNE COMPONENT.

Oral mucosa and skin are composed of highly specialized stratified epithelium that functions as a firstline barrier against physical and chemical damage. The integrity of this epithelial barrier is essentially dependent on structures maintaining cell-cell and cell-matrix adhesion .

Autoimmune bullous diseases are associated with autoantibodies directed against structures that mediate cell-cell and cellmatrix adhesion in skin and mucous membranes . In pemphigus diseases tissue injury is mediated by autoantibodies against the cell-cell junction causing intra-epithelial blistering, whereas in subepidermal autoimmune diseases autoantibodies are directed against the epithelial – connective tissue junction at the basement membrane zone (BMZ) .

Primary or extensive oral involvement is the hallmark of further inflammatory autoimmune conditions, including lichen planus (LP), erythema multiforme (EM), lupus erythematosus (LE) and chronic ulcerative stomatitis (CUS). Skin and oral mucosa are stratified epithelia, in which the cell-cell adhesion is mainly mediated by desmosomes and adherens junctions, whereas the adhesion of basal epithelial cells on the underlying basement membrane essentially depends on hemidesmosomes and focal contacts (Fig. 1).

Desmosomes are anchoring complexes that link epithelial cells to each other and attach the keratin filaments to the cell surface. Desmosomes consist of calcium-dependent adhesion molecules called cadherins, including desmogleins and desmocollins, which are transmembrane proteins that extend across the plasma membrane and mediate cell-cell adhesion by homo- or heterophilic interactions between their extracellular protein domains. An additional group of intracellular proteins resides on the cytoplasmic face of desmosomes and constitutes the desmosomal plaque.

Desmosomal plaque is associated with different types of proteins including plakoglobin, the desmoplakins, the plakophilins, envoplakin, and periplakin. It provides adhesion by linking the desmosomal transmembrane cadherin proteins to the cytoplasmic keratin filaments [1,5]. Hemidesmosomes are specialized junctional complexes on the ventral surface of the basal keratinocytes that maintain the epithelial cell attachment to the underlying basement membrane. In the oral cavity they can also be found in the junctional epithelium in contact to the tooth surface .

The basement membrane zone comprises the basal cell plasma membrane, the lamina lucida, the lamina densa and the sublamina densa. Anchoring filaments traverse the lamina lucida perpendicularly from the basal cell membrane to the underlying lamina densa.

At molecular levels, the basement membrane zone contains a mixture of structural components and antigens including collagen VII, which is the major structural component of anchoring fibrils, and collagen IV, which is a major ubiquitous component of vertebrate basement membranes. Laminins, which exist in various molecular forms as abundant non-collagenous glycoproteins of basement membranes, are heterotrimers consisting of alpha, beta and gamma chains.

Hemidesmosomes, together with the anchoring filaments, form the hemidesmosomes anchoring filament complex, which plays an important role in cell-basement membrane adhesion.

## TOPIC 26.

## CHANGES IN THE ORAL MUCOSA IN DERMATOSES WITH AN AUTOIMMUNE COMPONENT. RED LICHEN PLANUS. ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSIS. DENTIST'S TACTICS.

Lichen ruber planus (Lichen ruber planus) is the chronic disease, which is manifested on the skin and the mucous membranes by formation of the keratin- ized papules. LRP oral mucosa is usually encountered in people of middle age, predomi- nantly in women. Very rarely this illness can be found at younger age, and also in children.

Elements of lesion. Separate papules which appear on the skin are more often miliary. Their base is slightly infiltrated, round, oval, and sometimes elongated. Here at first the papules have lusterless colour, then they turn pink, reddish and even lilac-violet; sometimes longexisting papule turn brown. Since the papules are cornification, they slightly rise above the surrounding skin, The favourite places of the precipitations of papules LRP on the skin are flex- ible surfaces. On the hands – this is the region of radiocarpal joint. The development of disease is possible on mucosa membrane in different parts of gastrointestinal tract.

LRP of oral mucosa has predominantly two localizations: The most typical is the distal part of the cheek or the pear-shaped region, where the polygonal papules, after merging between themselves, form a figure in the form of fern leaves or grid; they are clearly defined and can be palpated. • The tongue (dorsal and lateral surface), in which the signs of disease are represented very diversely: the polygonal knots, which form an area either covered with scar-like strips as grids, the atrophic sections of oral mucosa, or hypertrophic papules.

Lichen planus – a common skin disease that is accompanied by lesions of the mucous membranes. Ill basically middle-aged adults majority of patients - women over 40 years. Particularly susceptible to the disease entity with excitable nervous system, as well as infected with hepatitis C. Lichen planus – idiopathic disease. We have the following hypotheses: a viral infection, autoimmunity, psychogenic reaction. The etiology and pathogenesis of the disease is unclear. It is believed that the basis for the development of the lichen planus are dysregulated immune and metabolic processes leading to inadequate tissue reaction under the influence of precipitating factors endogenous and exogenous origin. Available data suggest an important role of immunological disorders that result in T cells destroy the cells of the basal layer of the epithelium. Among the lymphocyte population submucosa identify CD4 and CD8. Lichen planus appearance on oral mucosa to some extent dependent on the presence of disease in patients with gastro-intestinal tract (gastritis, colitis, etc.), liver, pancreas. In some patients the relationship with vascular disease (hypertension) and endocrine (diabetes) disorders (Grynszpan's syndrome). A certain role in the development of the disease in the mucous membrane of the mouth is the latest injury, including those caused by dental pathology: the sharp edges of the teeth, bad pripasovannye removable plate dentures made of plastic, the absence of teeth, etc. Recently, there are increasing reports on the development of the lichen planus skin and oral mucosa in response to the effects on the body of certain chemicals, including pharmaceuticals. Describes the so-called lichenoid reactions in individuals who have contact with parafinilendiaminom taking tetracyclines (tetracyclines lichen), gold preparations, etc. In this way, the disease may in some cases be an allergic reaction to certain medications and chemical irritants. Lichen planus is a long disease with remissions and exacerbations. The clinic is characterized by the formation of a monomorphic rash, consisting of a flat, polygonal, with a shiny surface, and the retraction of papules with central pinkishpurple or crimson-red color, with a diameter of 2-3 mm. The papules may coalesce to form a small plaque on the surface of which there are small flakes. Small whitish points intertwined in a web, or lace fern

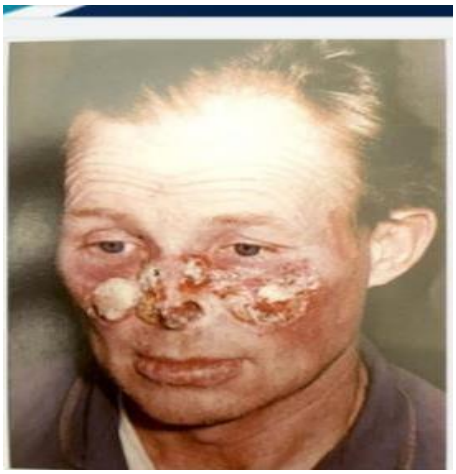
leaf, to shine through the stratum corneum (grid Wickham), due to the uneven thickening of the granular layer of the epidermis. At a resolution of lesions is often resistant hyperpigmentation. Lichen planus is accompanied by itching, often very intense, depriving patients of rest and sleep. Favourite localization planus – flexor surface of the forearm, wrist joints area, inner thighs and extensor – legs, inguinal and axillary regions, oral mucosa. Skin of the face, scalp, palms, soles are usually not involved in the process. Sometimes the rash is linear location, location is more often found in the limbs. In some patients, there is a pronounced change in nails with longitudinal striations, sometimes in the form of scallops, hyperemia of the nail bed with focal opacity of the nail plate of the hands and feet. For dermatosis lichen planus typical isomorphic response to stimulation. Often, the typical elements of dermatosis are linearly on the ground excoriations (phenomenon of Koebner). In 25% of patients defeat oral mucosa is not accompanied by manifestations in the skin. Lesions in the form of a typical localized symmetrically on the distal parts of the cheeks, in the retromolar region, on the surface of the tongue - flat lesions resembling leukoplakia, whitish with sharp jagged edges on the red border of lips (usually lower) – small purple patches, slightly scaly, having to surface grayish-white grid. On the oral mucosa are several atypical forms of the dermatosis lichen planus: When exudative form of congestive papules located on the background of edema and hyperemia oral mucosa, due to the clarity of the picture than smoothed. Patients complain of pain when used hot, acute and roughage, may be paresthesia and burning. Erosive and ulcerative form appears on the mucous membrane, and is the most severe, difficult to treat. In this form of the mucous membranes of the mouth or lips include erosion, ulceration least around which on hyperemic and edematous base arranged in a predetermined pattern typical lichen planus papules. Erosion have irregular shape, covered with fibrinous coating, after the removal of which easily leads to bleeding. Erosion can be kept for a long time, sometimes for years. Under the influence of the treatment erosion epiteliziruyutsya partially or completely, but recur at the same or another portion of the mucous membrane, sometimes immediately after cessation of treatment. Bullous shape - a rare exudative form, which is characterized by the formation of nodules near the typical vesicles with serous or sero-bloody contents. The quantity of bubbles of a pea or cherry, they quickly burst. Emerging erosion quickly epitelization. Patients complain of the occurrence of severe pain from eating any food, burning sensation. Hypertrophic, warty warty form is the result of hyperplasia. Manifested in the form of plaques purple or brownish-red in color, covered with warty hyperkeratotic stratifications. Around them are located some typical papules. Atypical form of lesions are located on the upper lip and gums. When placed on the lip lesion is symmetrical and is limited congestive hyperemia with clouding of the epithelium in the form of a whitish bloom. Papilla bloodshot, swollen, on the surface – a gentle off-white mesh. In typical cases, the diagnosis is not difficult. In case of difficulties in diagnosis, use additional methods of research. Histological examination helps to differentiate malignant from lichen planus. When studying the manifestations keratosis used fluorescent diagnosis. Differential diagnosis: pemphigus, syphilis, leukoplakia, lupus erythematosus, erythema multiforme, stomatitis (thrush, allergic, ulcerations, traumatic, necrotic Vincent); syndrome Grynszpan, a typical, exudative form hyperemic, erosive and ulcerative form, bullous, hypertrophic form, atypical form. Treatment of autoimmune dermatoses immunomodulation based on the principle (to change or correction of the immune response). The basic principle is to avoid the formation of autoantibodies. Therapy should be tailored to each individual case. Some types of autoimmune dermatoses are often not treated at all. Finally, use of the least toxic drug combination favoring drugs allowing to reduce side effects for a long time. It should not only be treated, but also take care to exclude factors directly worsen the patient's condition or contributing to the deterioration (such as certain autoimmune dermatoses light-sensitive or drug origin). Tactics dentist: the elimination of actinic keratoses, the normalization process of keratinization, eliminating inflammation, epithelialization defects.

Treatment. The complex therapy LRP includes two groups of the measures:

1. general action;
2. action directed to the LRP centre.

## TOPIC 27.

## CHANGES IN THE ORAL MUCOSA IN DERMATOSES WITH AN AUTOIMMUNE COMPONENT. RED LUPUS. DENTIST'S TACTICS.



An autoimmune disease that develops as a result of combined disorders of the immune system that lead to a chronic inflammatory process in many tissues and organs. The etiology is unknown.

The disease usually begins slowly, with the appearance of new symptoms over a period of weeks, months or even years. The most common first symptoms of SLE in children are nonspecific complaints of fatigue and malaise. Many children with SLE have recurrent or persistent fever, weight loss, and loss of appetite.

Over time, many children develop specific symptoms due to the involvement of one or more organs in the disease. Lesions of the skin and mucous membranes occur very often.

It can manifest itself in the form of various types of skin rash, photosensitivity (when exposure to the sun causes a rash), ulcers in the nose or mouth. A typical "butterfly" rash on the wings of the nose and cheeks occurs in one-third to one-half of sick children. In some cases, there is increased hair loss (alopecia). Hands may first turn red, then white and blue if the child is cold (Raynaud's syndrome).
Symptoms can also include swelling and joint pain, muscle aches, anemia, mild cyanosis, headaches, cramps, and chest pain. Kidney damage is present to one degree or another in most children, and largely determines the outcome of the disease over time. The most common symptoms of severe kidney damage are high blood pressure, blood and protein in the urine, swelling of the feet, legs, eyelids.

Lupus erythematosus can take several forms. Allocate: chronic (focal, limited, discoid form) and acute (systemic). Chronic focal lupus erythematosus is more often localized on the scalp and on the red border of the lower lip. There are 4 forms of it: 1) typical, 2) without clinically pronounced atrophy and hyperkeratosis, 3) erosive-ulcerative, 4) deep. The typical form is characterized by lip infiltration, the affected surface is dry, crimson-red, with persistently dilated vessels and a pronounced infiltration, due to which the lip is somewhat thickened and swollen. The lesions are located in the form of a tape, covered with tightly sitting scales, with the forcible removal of which bleeding and pain appear. In the center of the lesion, a focus of atrophy is observed. Histologically,

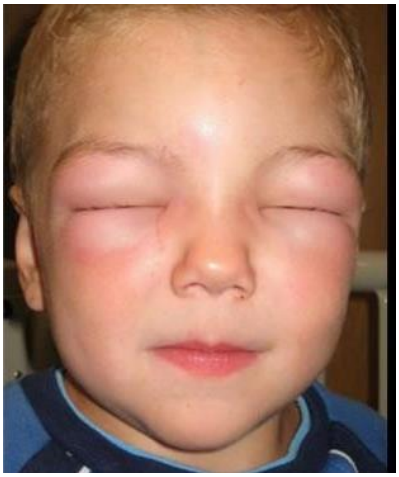
in the epithelium, hyperkeratosis, acanthosis, and in places clearly expressed atrophy are determined. The form of lupus erythematosus without clinically pronounced atrophy is characterized by diffuse congestive hyperemia and infiltration of the red border of the lips. With the erosive and ulcerative form of lupus erythematosus, there is a pronounced inflammation of the lips with erosions and cracks covered with crusts, as well as foci of hyperkeratosis. With deep formerythema and hyperkeratosis of the red border of the lips predominates. Systemic lupus erythematosus occurs with severe lesions visceral organs. In the clinical analysis of blood, leukopenia and an accumulation of lupus erythematosus cells with rosettes (LE cells) are observed. Treatment of lupus erythematosus includes: the appointment of synthetic antimalarial drugs (chloroquine diphosphate, delagil, rezohin) in combination with small doses of corticosteroid drugs (prednisolone, dexamethasone) and B vitamins, nicotinic acid. The daily dose of chloroquine and delagil is 0.25x 2 times a day, the course dose is 20.0. Lupus erythematosus foci should be protected from sunlight, strong 4 wind, rain, and injury. Topically applied ointment "Sinalar", "Lokakorten", prednisone, photoprotective creams.

**TOPIC 28**

**ACADEMIC HISTORY**

**TOPIC 29.**

# ANAPHYLACTIC SHOCK. SWELLING OF QUINCKE. CAUSES, CLINICAL MANIFESTATIONS, EMERGENCY CARE. ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSIS, TREATMENT AND PREVENTION. ALLERGIC MEDICATION STOMATITIS.



For some people with severe allergies, when they're exposed to something they're allergic to, they may experience a potentially life-threatening reaction called anaphylaxis. As a result, their immune system releases chemicals that flood the body. This can lead to anaphylactic shock.

Symptoms of anaphylaxis include:

skin reactions such as hives, flushed skin, or paleness

suddenly feeling too warm

feeling like you have a lump in your throat or difficulty swallowing

nausea, vomiting, or diarrhea

abdominal pain

a weak and rapid pulse

runny nose and sneezing

swollen tongue or lips

wheezing or difficulty breathing

a sense that something is wrong with your body

tingling hands, feet, mouth, or scalp

If you think you're experiencing anaphylaxis, seek medical attention immediately. If anaphylaxis has progressed to anaphylactic shock, the symptoms include:

struggling to breathe

dizziness

confusion

sudden feeling of weakness

loss of consciousness

First aid:

If someone appears to be going into anaphylactic shock, call emergency and then:

Get them into a comfortable position and elevate their legs. This keeps blood flowing to the vital organs.

Use EpiPen (epinephrine or adrenaline), administer it immediately.

Give CPR if patiend aren’t breathing until the emergency medical team arrives.

The first step for treating anaphylactic shock will likely be injecting epinephrine (adrenaline) immediately. This can reduce the severity of the allergic reaction.

At the hospital, receive more epinephrine intravenously (through an IV). You may also receive glucocorticoid and antihistamines intravenously. These medications help to reduce inflammation in the air passages, improving your ability to breathe.

Beta-agonists such as albuterol to make breathing easier can be given. You may also receive supplemental oxygen to help your body get the oxygen it needs.

Recognize patients with Quincke's edema will not be difficult - They "give" a swollen face: slit-like eyes, lips, cheeks. At hypostasis of internals pain, nausea, vomiting is observed. If the swelling has spread to the meninges, possible headache, dizziness, convulsions, loss of consciousness

Causes of edema:

-Acute allergic reaction to: food, plant pollen, animal hair, insect bites, dust, chemicals;

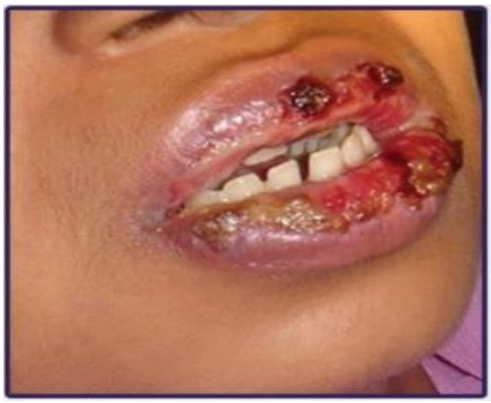
-The body's response to certain medications;

-Hereditary edema is a rare genetic disease in which allergic reactions in the body occur due to a lack of a special protein

Treatment: glucocorticoid and antihistamines intravenously, eufilin or any beta-agonists such as albuterol to make breathing easier can be given.

## TOPIC 30.

## MULTIFORME EXUDATIVE ERYTHEMA. STEVENS-JOHNSON SYNDROME. ETIOLOGY, PATHOGENESIS, CLINICAL MANIFESTATIONS, DIAGNOSIS, TREATMENT AND PREVENTION.



Exudative erythema multiforme is an acute agent characterized by polymorphic rashes on the skin and mucous membranes, cyclical course and tendency to recurrence, mainly in autumn.

Treatment program:

-At detection of the centers of a local infection their treatment is obligatory;

-Detoxification therapy (polyvidone, sodium chloride, potassium chloride, calcium chloride, magnesium chloride, sodium bicarbonate);

-Glucocorticoid drugs (in severe form - prednisolone, dexamethasone, triamcinolone, betamethasone);

External therapy: opening of large blisters followed by treatment with aniline dyes (fucorsin or methylthioninium chloride, etc.).

Stevens-Johnson syndrome is a severe immunocomplex disease characterized by the formation of blisters on the mucous membranes of the mouth, throat, eyes, genitals and other areas of the skin and mucous membranes caused by the body's response to various factors of biological or chemical origin

The causes of Stevens - Johnson syndrome are divided into four subgroups:

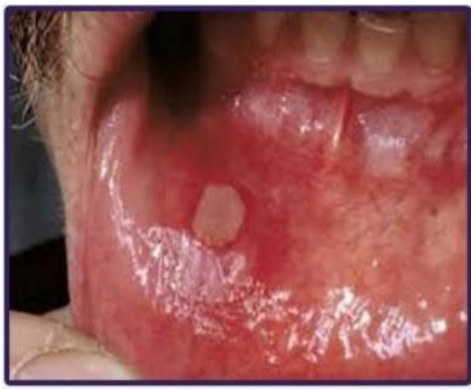
-Allergic reaction to drugs.

-Infections.

-The most common causes of Stevens-Johnson syndrome among viruses are herpesviruses, measles, viral hepatitis, mumps, and some SARS. Cancer. The most common causes of Stevens-Johnson syndrome among cancers are carcinomas and lymphomas.

## TOPIC 31.

## CHRONIC RECURRENT APHTHOUS STOMATITIS. BEHCET'S SYNDROME. PATHOGENESIS, CLINIC, DIAGNOSIS, TREATMENT AND PREVENTION.



Diagnostic criteria:

Clinical:

- soreness in the mouth;

- regional lymphadenitis;

- erosion (aphthae) of round or oval shape surrounded by a red border (1-3);

Laboratory:

- microbiological (detection of opportunistic and pathogenic microflora on the oral mucosa);

Light form:

- recurrences of aphthae occur once every few years.

Moderate form:

- recurrences of aphthae occur 1 - 3 times a year.

Severe form:

- recurrences of aphthae occur 4 or more times a year;

- aphthae turns into an ulcer.

Treatment:

General:

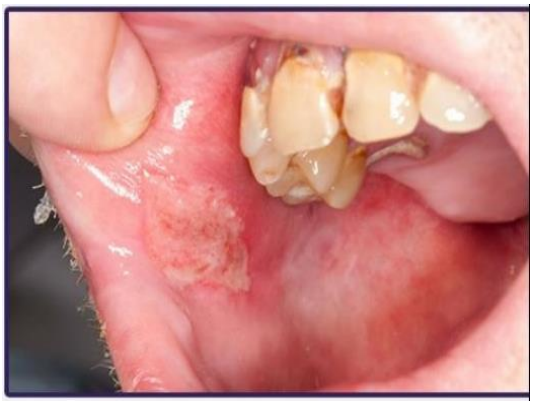
- immunomodulators (in moderate and severe forms of the disease);

- vitamins A, B, folic acid, and other drugs that stimulate epithelization (topically), iron preparations according to the indications

Local:

- anesthesia (local);

- antiseptics (locally);



Behçet's syndrome (often Behçet's disease) is a chronic autoimmune disease manifested by aphthous stomatitis, damage to the mucous membranes of the genitals, eyes (iritis, uveitis, and sometimes blindness) and often involvement of internal organs.

Reason

There are two hypotheses of the disease:

The first involves a genetic predisposition to the disease,

The second is the autoimmune hypothesis, confirmed by the presence of circulating immune complexes in the blood of patients, which accumulate in the tissues and cause their damage.

Treatment

Carried out tonics, desensitizing agents, if necessary - glucocorticosteroids. Topically, the oral cavity is rehabilitated in the interrecurrent period, applications of aphthous elements of enzyme preparations, analgesics, keratoplastic ointments are performed. In the presence of neurological symptoms, sedative therapy is indicated.

## TOPIC 32.

## CHANGES IN THE ORAL MUCOSA DURING EXOGENOUS INTOXICATION. DIAGNOSIS. DENTIST'S TACTICS.

Mercury stomatitis (stomatitis mercurialis) develops in people in whom mercury enters in the form of vapors through the respiratory tract, digestive tract, skin or by injection of mercury drugs for medical purposes. Metallic mercury does not have a toxic effect on the body in direct contact. Mercury stomatitis develops due to impaired tissue trophism associated with damage to the capillaries of the SOPR.

Clinic. The first symptom of mercury stomatitis is increased salivation, which is associated with irritation of the salivary glands secreted by mercury. The patient complains of a metallic taste in the mouth, a feeling of heat, severe throbbing pain in the gums, headache. The gums are inflamed, hyperemic, swollen. A gray-black border appears on the edge of the gums and gingival papillae, followed by a grayish-white foul-smelling plaque consisting of non-aerated epithelial cells and detritus. At deepening of necrotic process ulcers are formed, exposure between alveolar partitions is possible, often their sequestration, loosening and loss of teeth.

Treatment. Immediate cessation of mercury in the body, as well as measures to urgently remove it from the body. For this purpose appoint warm baths, alkaline mineral waters. The removal of mercury from the body is facilitated by the use of potassium iodide, sodium thiosulfate. Prescribe injections of unithiol, which binds mercury and forms with it insoluble compounds that are indifferent to the body. They are well excreted in urine and saliva.

## TOPIC 33.

## DAMAGE TO THE ORAL MUCOSA IN RADIATION DISEASE.

Possible side effects of radiation therapy. Radiation of the mouth and throat area can cause several short-term side effects, including:

Skin changes like a sunburn or suntan in the treated area that slowly fades away

Hoarseness

Loss of sense of taste

Redness and soreness or even pain in the mouth and throat

Sometimes open sores develop in the mouth and throat, making it hard to eat and drink during treatment. Liquid feeding through a tube placed into the stomach may be needed. (See Surgery for Oral Cavity and Oropharyngeal Cancer for more on tube feedings.) Radiotherapy may also cause long-lasting or permanent side effects:

Damage to the salivary glands: Permanent damage to the salivary (spit) glands can cause a dry mouth. This can lead to problems eating and swallowing.

The lack of saliva can also lead to tooth decay (cavities). People treated with radiation to the mouth or neck need to practice careful oral hygiene to help prevent this problem. Fluoride treatments may also help.

Newer radiotherapy techniques such as IMRT may help reduce this side effect. A drug called amifostine (Ethyol®) can also help reduce this side effect by limiting radiation damage to normal tissues. It's given into a vein over 15 minutes just before each radiation treatment. Amifostine has side effects, such as low blood pressure, nausea, and vomiting, that can make it hard to tolerate.

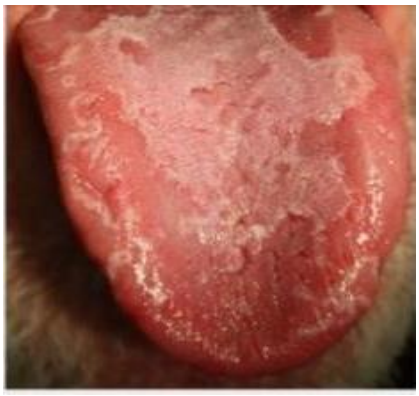
Damage to the jaw bone: This problem, known as osteoradionecrosis of the jaw, can be a serious side effect of radiation treatment. This is more common after tooth infection, extraction, or trauma, and it can be hard to treat. The main symptom is pain in the jaw. In some cases, the bone actually breaks. Sometimes the fractured bone heals by itself, but often the damaged bone will have to be repaired with surgery. To help prevent this problem, people getting radiation to the mouth or throat area need to see a dentist to have any problems with their teeth treated before radiation is started. In some cases, teeth may need to be removed.

## TOPIC 34.

## ACADEMIC HISTORY CONTROL

## TOPIC 35.

## PRIMARY GLOSSITIS. DESQUAMATIVE AND DIAMOND-SHAPED GLOSSITIS. FOLDED AND HAIRY TONGUE. CLINIC, DIAGNOSIS, TREATMENT AND PREVENTION.



The cause of geographical language can be a number of diseases:

-Diseases of the gastrointestinal tract;

-Disorders of B vitamins (B1, B2, B6, B12);

-Diseases of the stomach and duodenum (gastritis, gastroduodenitis, peptic ulcer);

-Malabsorption syndrome (violation of food absorption in the small intestine);

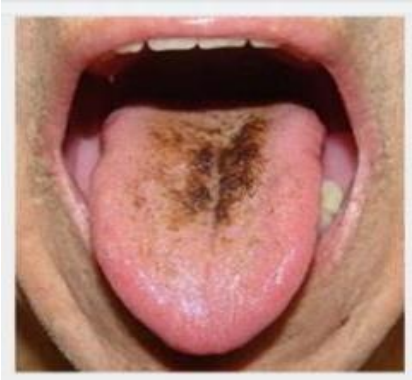
-Liver disease;

-Diseases of the pancreas (pancreatitis, diabetes, tumors;

-Some pathologies of the endocrine system (diseases of the thyroid gland, pancreas, adrenal glands);

Symptoms

The process usually begins with the appearance on the tongue of a gray area of turbidity with a diameter of several millimeters, which eventually swells, and in the center of it peel off filamentous papillae, exposing the red area of rounded shape. The peeling zone increases rapidly, while maintaining a level rounded shape. The intensity of desquamation (exfoliation) decreases. Desquamation spots can be of different sizes and shapes. Sometimes they have the shape of rings and semi-rings. in the zone of desquamation mushroom-shaped papillae in the form of bright red points are well visible



Causes of black tongue The disease, as mentioned earlier, occurs most often in middle-aged and older men. In this regard, we can identify specific favorable factors that can provoke the following disease:

Smoking abuse.

Tobacco smoking has a particularly strong effect.

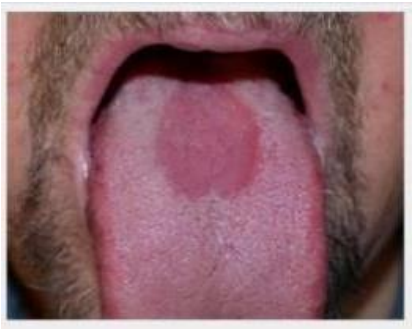
Abuse of coffee and strong tea.

Other reasons: Violations of oral hygiene, poor care of teeth and tongue. The use of some drugs, namely - drugs containing bismuth, as well as taking antibiotics. Water imbalance, dehydration, hyposalivation - insufficient amount of saliva. Irradiation of the head.

Treatment of black tongue

The main thing in the treatment of this disease is to restore oral hygiene and carefully monitor cleanliness. Brush your teeth at least twice a day, preferably after each meal.

Also, the patient should increase the amount of fluid consumed and give up bad habits and drinking coffee and tea. If the development of black tongue is caused by fungi or bacteria, the doctor should prescribe appropriate courses of antibiotics or antifungal drugs.



Symptoms and signs

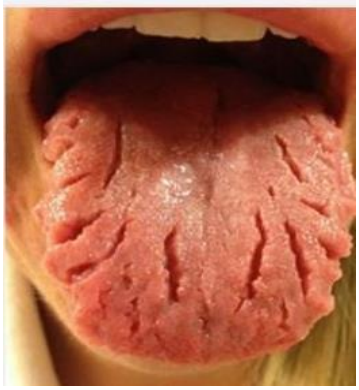
The external manifestations of rhomboid glossitis of the tongue depend on the form of the disease, but the general symptoms are characteristic of all types of glossitis.

At the initial stage, the pathology manifests itself only in the form of lesions of the back of the tongue: the formation in the form of a diamond or oval of bluish-red hue has a length of not more than 2 centimeters and a width of not more than 5 centimeters.

The middle form is accompanied by a feeling of a foreign body in the mouth, swelling of the tongue, increased salivation. Diction disorders are possible.

As the disease progresses, the symptoms worsen: ulcers appear, taste sensations disappear. The patient has difficulty talking, eating and even drinking, at the slightest impact there is a sharp piercing pain.

Treatment of the smooth form does not involve surgery, only the appointment of the necessary drugs. At bumpy and papillomatous type the acting fabrics are cut off. To begin with, the patient undergoes complete rehabilitation of the oral cavity - removes plaque, tartar, caries and other pathologies to eliminate pathogenic bacteria. Then prescribe antifungal and antiseptic drugs, a course of vitamins, healing ointments. If the reason was a disorder of the stomach, a special diet is recommended.



Folded (scrotal) tongue - a congenital anomaly of the shape and size of the tongue, which is expressed in the presence of deep grooves (folds) running in different directions.

Symptoms

Folding is often accompanied by a moderate increase in the whole language - macroglossia. The presence of numerous furrows on its surface is characteristic. The longitudinal fold is usually located strictly in the middle, originating from the tip of the tongue and often reaching the level of the location of the grooved papillae, from it depart transverse folds (in the form of leaf veins).

## TOPIC 36.

## NEUROGENIC DISEASES OF THE TONGUE. ETIOLOGY, PATHOGENESIS, CLINIC, DIAGNOSIS, TREATMENT AND PREVENTION.

Melkersson–Rosenthal syndrome (also termed "Miescher-MelkerssonRosenthal syndrome"),[1] is a rare neurological disorder characterized by recurring facial paralysis, swelling of the face and lips (usually the upper lip), and the development of folds and furrows in the tongue.[2]:799 Onset is in childhood or early adolescence. After recurrent attacks (ranging from days to years in between), swelling may persist and increase, eventually becoming permanent. The lip may become hard, cracked, and fissured with a reddish-brown discoloration. The cause of Melkersson–Rosenthal syndrome is unknown, but there may be a genetic predisposition. It has been noted to be especially prevalent among certain ethnic groups in Bolivia. It can be symptomatic of Crohn's disease or sarcoidosis.Treatment

Treatment is symptomatic and may include nonsteroidal antiinflammatory drugs (NSAIDs) and corticosteroids to reduce swelling, antibiotics and immunosuppressants. Surgery may be indicated to relieve pressure on the facial nerves and reduce swelling, but its efficacy is uncertain. Massage and electrical stimulation may also be prescribed.

Melkersson–Rosenthal syndrome may recur intermittently after its first appearance. It can become a chronic disorder. Follow-up care should exclude the development of Crohn's disease or sarcoidosis.

## TOPIC 37.

## CHEILITES. ETIOLOGY, CLINIC, DIAGNOSIS, TREATMENT AND PREVENTION.

| | |
|---|---|
| clinical manifestations , treatment and prognosis of meteorological cheilitis | Mashkill l Eyson AL was first described as an independent disease . Ill mostly men aged 20-75, the okiv , working in adverse weather conditions. Red border , preferably lower lip , not clearly hyperemic, infiltrated, dry, covered with small scales. CO lips without changes. The course is chronic and depends on the time of year. . **Histologically:** diffuse irregular epithelial hyperplasia, sometimes with a slight keratinization. |
| | **Treatment:** weaken the effect of meteofactors , locally anti-inflammatory ointments , for general treatment - vitamins of group B2 , B6, B12 and oil solution of vitamin A 10 drops 3 times a day for a month.<br>**Prognosis** : favorable when the patient is less in the open air. In the long run, malignancy is possible. |
| clinical manifestations , treatment and prognosis of actinic cheilitis | This is a chronic disease caused by the increased sensitivity of the red lip border to sunlight, resulting in a delayed type allergic reaction. First written by S. Aeres in 1923 . More often men 20-60 years get sick.<br>**Dry form:** hot lips, red border of lower lip bright red, covered with dry, small, silvery-white scales. Once removed, they reappear. The red lip of the lips is rough, easily injured. In the long run it is possible to develop cracks, erosion.<br>**The exudative form is** characterized by acute inflammatory phenomena: pain, red border edging, hyperemia, lip swelling, blisters, erosion, painful cracks, crusts. A characteristic feature of actinic cheilitis is the absence of damage to the corners of the mouth.<br>**Treatment:** protection of lips from sunlight , antihistamines, imudon, applications of anti-inflammatory ointments , keratoplasty.<br>**Prediction :** 1% of cases emerged and is malignancy of the lips . Active observation and timely treatment makes the prognosis favorable. |

| | |
|---|---|
| clinical manifestations , treatment and prognosis of contact allergic cheilitis | Contact allergic second cheilitis first described Miller - Taussing in 1925. The vast majority of patients are women aged 20-60 years. The disease occurs under the action of chemicals that are components of lipstick, components of toothpastes, powders, plastics of dentures. The disease can also be professional.<br>Characteristic is hotness, itching, mild pain, hyperemia, swelling, blistering, erosion, wetting. In milder cases, there is slight hyperemia, dryness, tightness of the lips, flaking, slight edema of the red lip border , cracks on the border with the skin. Provocative test (effect of elimination) - 5-7 days after the disappearance of acute events repeated use of a single factor , cause relapse.<br>**Treatment:** abolishing the use of the factors that led to the appearance of cheilitis, the application of anti-inflammatory ointments. General treatment: antihistamines.<br>**The prognosis is** favorable if allergen contact is not allowed again. |
| clinical manifestations , treatment and prognosis of of chronic lip fracture | On the lower lip poodyn exhibit at mo , linear defects of varying depth, length 1.5 cm, sometimes covered with bloody crust. Cracks can pass to the mucous membrane of the lip. Cracking without treatment is long-lasting (up to 3-8 years), with alternation of remissions and relapses. T rischyny hlybshayut formed painful infiltration, the edges are sealed, cell hyperkeratosis<br>. **Histologically:** chronic inflammation that is accompanied |
| | with the regenerative, hyperplastic, and sometimes metaplastic epithelial growth .<br>**Treatment:** to conservative and surgical . Conservative - AE Vit, a complex of B vitamins internally. Topical use of keratoplastics, antimicrobials, anti-inflammatory ointments in combination with antibiotics, adhesive dressings , blockages 0.25 - 0.5 ml of 1% novocaine, helium-neon laser. Surgical - cryodestruction.<br>**The prognosis is** favorable, but with long-term development of precancerous diseases (leukoplakia, skin horn) is possible. |

Meteorological cheilitis ( cheilitis meteorologic a) was first described as an independent disease by ALMashkillayson. Diagnosis of cheilitis is complex and requires a differential diagnosis with contact allergic, atopic, exfoliative (dry form), actinic, lupus erythematosus and red spotted lichen. The prognosis is favorable when the patient is less in the open air or changes his place of work, the signs of the disease diminish and even disappear. In the long run it becomes a background for the development of precancerous diseases, malignancy is possible.

Treatment :

- it is not necessary to reduce the influence of factors that caused the disease (hygienic lipstick, creams "Lux", "Delight", "Spermacetic")

- for general treatment, prescribe vitamins B 2 , B 6 , B 12 , nicotinic acid and oil vitamin A 10 drops 3 times a day for a month

- and corticosteroid-based ointments (senator and others)

- For the correction of the immune system - Imudone, as well as IgA in tablets under the tongue for resorption 7- 8 times a day.

Actinic cheilitis (cheilitis actinica) is a chronic condition that is caused by the increased sensitivity of the red lip border to sunlight. First described by S. Ayres in 1923. Differential diagnosis is required with exfoliative cheilitis, contact allergic and atopic cheilitis.

Treatment :

- protection of lips from sunlight (creams "Ray", "Shield", aerosol "Fencortozol")

- chingamine 0.25 1 time a day, starting in the spring, gradually increasing the dose to 2-3 tablets a day

- sIgA under the tongue before resorption (8 times a day )

- application of ointment fluoro for Horta ketaloha, sinalara, lokortena and others.

Actinic cheilitis is the background for the occurrence of precancerous malignancies. Hence the need for active surveillance, timely treatment, which makes the prognosis favorable.

Contact allergic cheilitis ( cheilitis venenata , s cheilitis allergica contactilis ). First described in 1925 by Miller - Taussing . Diagnosis is based on a clear identification of allergic history. The setting of skin tests is complicated because the skin reacts differently than the red border. What matters is the provocative test (elimination effect): 5-7 days after the disappearance of acute effects, repeated use of lipstick causes recurrence. Differential diagnosis should be made with actinic, meteorological, exfoliative, eczematous and atopic cheilitis.

Treatment . The use of the factors that led to cheilitis, the administration of corticosteroid ointment applications (senator, canine, etc.) 5-6 times a day is abolished. With a significant inflammatory reaction, prescribe intra-antihistamines (fincarol, clarithin, etc.). The prognosis is favorable if allergen contact is not allowed again.

Glandular cheilitis ( cheilitis glandularis ) is an inflammatory disease of the displaced into the transitional zone (Klein zone) and sometimes into the red border, small glands. He first introduced the term into Volkman's medical literature in 1870. Diagnosis is not difficult due to the peculiar clinic. Differential diagnosis , depending on the form, should be made with secondary forms of glundular cheilitis occurring on the background of red spotted lichen, leukoplakia, tuberculous and red lupus, with polycystosis, with lymphoedematous and granulomatous cheilitis. The prognosis for glondular cheilitis is favorable. But it should be remembered that there may be a precancerous disease of red lip border.

Treatment

is surgical and conservative.

In inflammatory processes:

- corticosteroid ointments with antibiotics (Si setting H, lococorten H, hyoxyzone)

- in purulent inflammation, antibiotics per os and topically (5%, 10% sintomitsinovoy emulsion fulevyl, Levosin 3% tetratsyk linova eritromitsynova or ointment)

- correction of the immune system: IIU forth on, sIgA.

Chronic cleft lip (rhagas labia chronica) is a limited inflammation that results in a slit-like linear defect in the epithelium and its own plate.

Diagnosis of chronic cleft lip is not complicated. The prognosis is favorable, but over the long-term it becomes the background for the development of precancerous diseases (leukoplakia, skin horn). Treatment of chronic cracks is conservative and surgical. For conservative therapy use vitamin preparations (aevit, a complex of B vitamins), which are administered per os. In lesions use keratoplasty (retinol, aevit, rosehip oil, sea buckthorn, methyluracil ointment), antimicrobials (10% syntomycin emulsion, levovinisol, levomekol, etc.), corticosteroid ointments in combination with dibiotic, antibiotic, corticosterol, antibiotics dressings based on biological glue, as well as the blockade of 0.25-0.5 ml of 1% novocaine in the base of the crack on the oral side - 1-2 blockades with an interval of 5-7 days, heliumneon laser. In the conditions of appearance at cracks of scar atrophy of its edges or hyperkeratosis surgical removal of the lesion is shown.

Exfoliative cheilitis ( cheilitis exfoliativa ). It was first described by Stelwagou (1900) under the name "transient lip desquamation". Mikulicz and K ü mmel (1912) named it exfoliative cheilitis. It is an xudative form. Diagnosis is usually not difficult: typical localization of the lesion, no erosion, characteristic appearance of the crusts. Differential diagnostics should be performed with eczematous, actinic, abrasive precancerous Manganotti cheilitis, erosive-ulcerative form of red spotted lichen and lupus erythematosus, vulgar blistering, multiform exudative erythema. Dry form . Differential diagnosis should be made with meteorological, atopic, actinic (dry form) and atrophic fungal cheilitis.

Treatment. General treatment (should be performed with an endocrinologist, psychoneurologist):

- sedatives (infusion of valerian, hermit, peony);
- tranquilizers (Sebazon, Phenozepam, Eleniums, Mezapam);
- in severe depressive states - antidepressant drugs (amitriptyline, azafen);
- antihistamines (fencorol, clarithin);
- vitamins C and group B in therapeutic doses;
- Immune and sIgA in tablets up to 8 times a day are prescribed for the immune system to be corrected, kept in the mouth until absorbed.

Locally in the lesion area use :

- corticosteroid ointments (triacort, fluorocort, senator, canalologist);
- in the presence of microbial flora - hormonal ointments with antibiotics

(dermozolone, dexocort, corticomycetin, fencortisol).

In case of ineffectiveness of conservative therapy, Bucca beams are prescribed: 1 Gy - 1 time per week up to 2-3 Gy at intervals of 7-10 days. Course dose from 10 to 12-20 Gy.

Eczema cheilitis (cheilitis exzematosa) - a chronic recurrent allergic disease of red border and lip skin. There are isolated, eczematous lesions of the lips, but more often it is a symptom of skin eczema. Diagnosis is facilitated by the fact that there is a classic skin lesion. In other cases, the diagnosis of eczematous cheilitis is based on the presence of microvesicles, point-like serous wells, evolutionary polymorphism of lesions. Differential diagnosis should be made with contact allergic, meteorological actinic, atopic, exfoliative cheilitis.

Treatment of eczematous cheilitis complex:

- sedative therapy;
- antihistamines;
- low doses of corticosteroids and antibiotics (with microbial eczema);
- immunocorrector immunoconduct, sIgA;
- Gintosuggestive therapy, electrosleep.

Local:

- corticosteroid ointments, aerosols (lococorten, flucinar, fluorocort, celestoderm, triderm).

Atopic cheilitis (cheilitis atopicalis) is a symptom of atopic dermatitis or neurodermatitis. Diagnosed more often in children 7-17 years. Diagnosis does not cause much difficulty. Attention should be paid to lesions of the skin of the neck, extremities (lesions of the hamstrings and elbows). Help to correctly diagnose changes in peripheral blood: lymphocytosis, eosinophilia, decrease in the number of E-lymphocytes, T-suppressors, increase in B-lymphocytes, hyperproduction of IgE. Differential diagnostics . Atopic cheilitis should be differentiated with exfoliative, contact allergic, meteorological, actinic, eczematous, mycotic, and streptococcal angular cheilitis. The outlook is favorable.

Treatment of atopic cheilitis is complex .

General :

- antihistamines;
- vitamins (B 2 , B 6 );
- tranquilizers (Elenium, Seduxen, Tazepam);
- hypo sensitizing histoglobulin therapy during the acute phase of the disease
- for the correction of the immune system: imudon, sIgA;

- for severe disease - a short course of corticosteroid therapy (prednisone, dexamethasone);
- diet (excluding salty, spicy, spicy foods, alcohol, reducing carbohydrates);
- spa treatment in dry and warm climates.

Local Interventions:

- corticosteroid ointments (fluorocort, senator, flucinar, betnovate, locolortene);
- for lichenization - application of 10-20% ichthyol ointment or 10% naphthylanov liniment;
- physical procedures: magnetotherapy, infrared rays, laser therapy.

## TOPIC 38.

## PRECANCEROUS DISEASES. CLASSIFICATION OF PRECANCERS OF THE ORAL MUCOSA AND RED LIP BORDER. THE PROGNOSIS AND PREVENTION OF PRECANCEROUS DISEASES.

Classification of pretumor processes of oral mucosa

With high frequency of malignization (obligate): 1) morbus Bowen.

With little frequency of malignization (facultative): 1) verrucuos and erosive leukokeratosis; 2) polypapilloma; 3) erosive ulcerous and hyperkeratotic forms of erythema centrifugum and lichen acuminatus; 4) postradial stomatitis.

Classification of pretumor processes of red border

With high frequency of malignization (obligate):

1) verrucous precarcinoma; 2) limited precancerous hyperkeratosis; 3) abrasiva praecancerosa cheilitis Manganotti.

With little frequency of malignization (facultative): 1) leukokeratosis; 2) kera-toacanthoma; 3) cutaneous horn; 4) papilloma with comification; 5) erosive ulcerous and hyperkeratotic forms of erythema centrifugum and lichen acuminatus; 6) postradial cheilitis.

Leukoplakia

Leukoplakia generally refers to a firmly attached white patch on a mucous membrane which is associated with an increased risk of cancer. The edges of the lesion are typically abrupt and the lesion changes with time. Advanced forms may develop red patches. There are generally no other symptoms. It usually occurs within the mouth, although sometimes mucosa in other parts of the gastrointestinal tract, urinary tract, or genitals may be affected.

Leukoplakia is a descriptive term that should only be applied after other possible causes are ruled out. Tissue biopsy generally shows increased keratin build up with or without abnormal cells, but is not diagnostic. Other conditions that can appear similar include yeast infections, lichen planus, and keratosis due to repeated minor trauma. The lesions from a yeast infection can typically be rubbed off while those of leukoplakia cannot.

Treatment recommendations depend on features of the lesion. If abnormal cells are present or the lesion is small surgical removal is often recommended; otherwise close follow up at three to six month intervals may be sufficient. People are generally advised to stop smoking and limit the drinking of alcohol. In potentially half of cases leukoplakia will shrink with stopping smoking; however, if smoking is continued up to 66% of cases will become more white and thick. The percentage of people affected is estimated at 1–3%. Leukoplakia becomes more common with age, typically not occurring until after 30. Rates may be as high as 8% in men over the age of 70.

Definition

The word leukoplakia means "white patch", and is derived from the Greek words λευκός - "white" and πλάξ - "plate". Leukoplakia is a diagnosis of exclusion, meaning that which lesions are included depends upon what diagnoses are currently considered acceptable. Accepted definitions of leukoplakia have changed over time and are still controversial. It is possible that the definition will be further revised as new knowledge becomes available.

In 1984 an international symposium agreed upon the following definition: "a whitish patch or plaque, which cannot be characterized clinically or pathologically as any other disease, and is not associated with any physical or chemical agent except the use of tobacco." There were however problems and confusion in applying this definition. At a second international symposium held in 1994 it was argued that whilst tobacco was a likely causative factor in the development of leukoplakia, some white patches could be linked directly to the local effects of tobacco by virtue of their disappearance following smoking cessation, suggesting that this kind of white patch represents a reactive lesion to local tissue irritation rather than a lesion caused by carcinogens in cigarette smoke, and could be better termed to reflect this etiology, e.g. smokers' keratosis. The second international symposium therefore revised the definition of leukoplakia to: "a predominantly white lesion of the oral mucosa that cannot be characterized as any other definable lesion."

In the mouth, the current definition of oral leukoplakia adopted by the World Health Organization is "white plaques of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer". However, this definition is inconsistently applied in the medical literature, and some refer to any oral white patch as "leukoplakia".

The term has been incorrectly used to describe white patches of any cause (rather than specifically referring to idiopathic white patches) and also to refer only to white patches which have a risk of cancerous changes. It has been suggested that leukoplakia is an unhelpful term since there is so much inconsistency surrounding its use, and some clinicians now avoid using it at all.

Classification

- Leukoplakia in the lower labial sulcus
- Leukoplakia of the soft palate
- Exophytic leukoplakia on the buccal mucosa
- Leukoplakia on the side of tongue

Leukoplakia could be classified as mucosal disease, and also as a premalignant condition. Although the white color in leukoplakia is a result of hyperkeratosis (or acanthosis), similarly appearing white lesions that are caused by reactive keratosis (smoker's keratosis or frictional keratosis e.g. morsicatio buccarum) are not considered to be leukoplakias. Leukoplakia could also be considered according to the affected site, e.g. oral leukoplakia, leukoplakia of the urinary tract, including bladder leukoplakia or leukoplakia of the penis, vulvae, cervix or vagina. Leukoplakia may also occur in the larynx, possibly in association with gastro-esophageal reflux disease. Oropharyngeal leukoplakia is linked to the development of esophageal squamous cell carcinoma, and sometimes this is associated with tylosis, which is thickening of the skin on the palms and soles of the feet (see: Leukoplakia with tylosis and esophageal carcinoma). Dyskeratosis congenital may be associated with leukoplakia of the oral mucosa and of the anal mucosa.

Mouth

Within the mouth, leukoplakia is sometimes further classified according to the site involved, e.g. leukoplakia buccalis (leukoplakia of the buccal mucosa) or leukoplakia lingualis (leukoplakia of the lingual mucosa). There are two main clinical variants of oral leukoplakia, namely homogenous leukoplakia and non-homogenous (heterogenous) leukoplakia, which are described below. The word leukoplakia is also included within the nomenclature of other oral conditions which present as white patches, however these are specific diagnoses which are generally considered separate from leukoplakia, with the notable exception of proliferative verrucous leukoplakia, which is a recognized sub-type of leukoplakia.

Homogenous leukoplakia

Homogenous leukoplakia (also termed "thick leukoplakia") is usually well defined white patch of uniform, flat appearance and texture, although there may be superficial irregularities. Homogenous leukoplakia is usually slightly elevated compared to surrounding mucosa, and often has a fissured, wrinkled or corrugated surface texture, with the texture generally consistent throughout the whole lesion. This term has no implications on the size of the lesion, which may be localized or extensive. When homogenous leukoplakia is palpated, it may feel leathery, dry, or like cracked mud.

Non-homogenous leukoplakia

Non-homogenous leukoplakia is a lesion of non-uniform appearance. The color may be predominantly white or a mixed white and red. The surface texture is irregular compared to homogenous leukoplakia, and may be flat (papular), nodular or exophytic. "Verrucous leukoplakia" (or "verruciform leukoplakia") is a descriptive term used for thick, white, papillary lesions. Verrucous leukoplakias are usually heavily keratinized and are often seen in elderly people. Some verrucous leukoplakias may have an exophytic growth pattern, and some may slowly invade surrounding mucosa, when the term proliferative verrucous leukoplakia may be used. Non-homogeneous leukoplakias have a greater risk of cancerous changes than homogeneous leukoplakias.

Proliferative verrucous leukoplakia

Proliferative verrucous leukoplakia (PVL) is a recognized high risk subtype of non-homogenous leukoplakia. It is uncommon, and usually involves the buccal mucosa and the gingiva (the gums). This condition is characterized by (usually) extensive, papillary or verrucoid keratotic plaques that tends to slowly enlarge into adjacent mucosal sites. An established PVL lesion is usually thick and exophytic (prominent), but initially it may be flat. Smoking does not seem to be as strongly related as it is to leukoplakia generally, and another dissimilarity is the preponderance for women over 50. There is a very high risk of dysplasia and transformation to OSCC or to verrucous carcinoma.

Erythroleukoplakia ("speckled leukoplakia"), left commissure. Biopsy showed mild epithelial dysplasia and candida infection. Antifungal medication may turn this type of lesion into a homogenous leukoplakia (i.e. the red areas would disappear)

Erythroleukoplakia (also termed speckled leukoplakia, erythroleukoplasia or leukoerythroplasia) is a non-homogenous lesion of mixed white (keratotic) and red (atrophic) color. Erythroplakia (erythroplasia) is an entirely red patch that cannot be attributed to any other cause. Erythroleukoplakia can therefore be considered a variant of either leukoplakia or erythroplakia since its appearance is midway between. Erythroleukoplakia frequently occurs on the buccal mucosa in the commissural area (just inside the cheek at the corners of the mouth) as a mixed lesion of white nodular patches on an erythematous background, although any part of the mouth may be affected. Erythroleukoplakia and erythroplakia have a higher risk of cancerous changes than homogeneous leukoplakia.

Signs and symptoms

Most cases of leukoplakia cause no symptoms, but infrequently there may be discomfort or pain. The exact appearance of the lesion is variable. Leukoplakia may be white, whitish yellow or grey. The size can range from a small area to much larger lesions. The most common sites affected are the buccal mucosa, the labial mucosa and the alveolar mucosa, although any mucosal surface in

the mouth may be involved. The clinical appearance, including the surface texture and color, may be homogenous or non-homogenous. Some signs are generally associated with a higher risk of cancerous changes.

Causes

The exact underlying cause of leukoplakia is largely unknown, but it is likely multifactorial, with the main factor being the use of tobacco. Tobacco use and other suggested causes are discussed below. The mechanism of the white appearance is thickening of the keratin layer, called hyperkeratosis. The abnormal keratin appears white when it becomes hydrated by saliva, and light reflects off the surface evenly. This hides the normal pink-red color of mucosae (the result of underlying vasculature showing through the epithelium). A similar situation can be seen on areas of thick skin such as the soles of the feet or the fingers after prolonged immersion in water. Another possible mechanism is thickening of the stratum spinosum, called acanthosis.

Tobacco

Tobacco smoking or chewing is the most common causative factor, with more than 80% of persons with leukoplakia having a positive smoking history. Smokers are much more likely to suffer from leukoplakia than non-smokers. The size and number of leukoplakia lesions in an individual is also correlated with the level of smoking and how long the habit has lasted for. Other sources argue that there is no evidence for a direct causative link between smoking and oral leukoplakia. Cigarette smoking may produce a diffuse leukoplakia of the buccal mucosa, lips, tongue and rarely the floor of mouth. Reverse smoking, where the lit end of the cigarette is held in the mouth is also associated with mucosal changes. Tobacco chewing, e.g. betel leaf and areca nut, called paan, tends to produce a distinctive white patch in a buccal sulcus termed "tobacco pouch keratosis". In the majority of persons, cessation triggers shrinkage or disappearance of the lesion, usually within the first year after stopping.

Alcohol

Although the synergistic effect of alcohol with smoking in the development of oral cancer is beyond doubt, there is no clear evidence that alcohol is involved in the development of leukoplakia, but it does appear to have some influence. Excessive use of a high alcohol containing mouth wash (> 25%) may cause a grey plaque to form on the buccal mucosa, but these lesions are not considered true leukoplakia.

Ultraviolet radiation

Ultraviolet radiation is believed to be a factor in the development of some leukoplakia lesions of the lower lip, usually in association with actinic cheilitis.

Micro-organisms

Candida in its pathogenic hyphal form is occasionally seen in biopsies of idiopathic leukoplakia. It is debated whether candida infection is a primary cause of leukoplakia with or without dysplasia, or a superimposed (secondary) infection that occurs after the development of the lesion. It is known that Candida species thrive in altered tissues. Some leukoplakias with dysplasia reduce or disappear entirely following use of antifungal medication. Smoking, which as discussed above can lead to the development of leukoplakia, can also promote oral candidiasis. Candida in association with leukoplakia should not be confused with white patches which are primarily caused by candida infection, such as chronic hyperplastic candidiasis ("candidal leukoplakia").

The involvement of viruses in the formation of some oral white lesions is well established, e.g. Epstein-Barr virus in oral hairy leukoplakia (which is not a true leukoplakia). Human papilloma virus (HPV), especially HPV 16 and 18, is sometimes found in areas of leukoplakia, however since this virus can be coincidentally found on normal, healthy mucosal surfaces in the mouth, it is unknown if this virus is involved in the development of some leukoplakias. In vitro experimentation has demonstrated that HPV 16 is capable of inducing dysplastic changes in previously normal squamous epithelium.

Epithelial atrophy

Leukoplakia is more likely to develop in areas of epithelial atrophy. Conditions associated with mucosal atrophy include iron deficiency, some vitamin deficiencies, oral submucous fibrosis, syphilis and sideropenic dysphagia.

Trauma

Another very common cause of white patches in the mouth is frictional or irritational trauma leading to keratosis. Examples include nicotine stomatitis, which is keratosis in response to heat from tobacco smoking (rather than a response to the carcinogens in tobacco smoke). The risk of malignant transformation is similar to normal mucosa. Mechanical trauma, e.g. caused by a sharp edge on a denture, or a broken tooth, may cause white patches which appear very similar to leukoplakia. However, these white patches represent a normal hyperkeratotic reaction, similar to a callus on the skin, and will resolve when the cause is removed. Where there is a demonstrable cause such as mechanical or thermal trauma, the term idiopathic leukoplakia should not be used.

Differential diagnosis

There are many known conditions which present with a white lesion of the oral mucosa, but the majority of oral white patches have no known cause. These are termed leukoplakia once other likely possibilities have been ruled out. There are also few recognized subtypes of leukoplakia, described according to the clinical appearance of the lesion.

Almost all oral white patches are usually the result of keratosis. For this reason oral white patches are sometimes generally described as keratoses, although a minority of oral white lesions are not related to hyperkeratosis, e.g. epithelial necrosis and ulceration caused by a chemical burn. In keratosis, the thickened keratin layer absorbs water from saliva in the mouth and appears white in comparison with normal mucosa. Normal oral mucosa is a red-pink color due the underlying vasculature in the lamina propria showing through the thin layer of epithelium. Melanin produced in the oral mucosa also influences the color, with a darker appearance being created by higher levels of melanin in the tissues (associated with racial/physiologic pigmentation, or with disorders causing melanin overproduction such as Addison's disease). Other endogenous pigments can be overproduced to influence the color, e.g. bilirubin in hyperbilirubinemia or hemosiderin in hemochromatosis, or exogenous pigments such as heavy metals can be introduced into the mucosa, e.g. in an amalgam tattoo.

Leukoplakia cannot be rubbed off the mucosa, distinguishing it readily from white patches such as pseudomembranous candidiasis, where a white layer can be removed to reveal an erythematous, sometimes bleeding surface underneath. The white color associated with leukoedema disappears when the mucosa is stretched. A frictional keratosis will generally be adjacent to a sharp surface such as a broken tooth or rough area on a denture and will disappear when the causative factor is removed. Some have a suggested as general rule that any lesion that does not show signs of healing within 2 weeks should be biopsied. Morsicatio buccarum and linea alba are located at the level of the occlusal plane (the level at which the teeth meet). A chemical burn has a clear history of

placing an aspirin tablet (or other caustic substance such as eugenol) against the mucosa in an attempt to relieve toothache. Developmental white patches usually are present from birth or become apparent earlier in life, whilst leukoplakia generally affects middle aged or elderly people. Other causes of white patches generally require pathologic examination of a biopsy specimen to distinguish with certainty from leukoplakia.

Management

A systematic review found that no treatments commonly used for leukoplakia have been shown to be effective in preventing malignant transformation. Some treatments may lead to healing of leukoplakia, but do not prevent relapse of the lesion or malignant change. Regardless of the treatment used, a diagnosis of leukoplakia almost always leads to a recommendation that possible causative factors such as smoking and alcohol consumption be stopped, and also involves long term review of the lesion, to detect any malignant change early and thereby improve the prognosis significantly.

Predisposing factors and review

Beyond advising smoking cessation, many clinicians will employ watchful waiting rather than intervene. Recommended recall intervals vary. One suggested program is every 3 months initially, and if there is no change in the lesion, then annual recall thereafter. Some clinicians use clinical photographs of the lesion to help demonstrate any changes between visits. Watchful waiting does not rule out the possibility of repeated biopsies. If the lesion changes in appearance repeat biopsies are especially indicated. Since smoking and alcohol consumption also places individuals at higher risk of tumors occurring in the respiratory tract and pharynx, "red flag" symptoms (e.g. hemoptysis - coughing blood) often trigger medical investigation by other specialties.

Surgery

Surgical removal of the lesion is the first choice of treatment for many clinicians. However, the efficacy of this treatment modality cannot be assessed due to insufficient available evidence. This can be carried out by traditional surgical excision with a scalpel, with lasers, or with eletrocautery or cryotherapy. Often if biopsy demonstrates moderate or severe dysplasia then the decision to excise them is taken more readily. Sometimes white patches are too large to remove completely and instead they are monitored closely. Even if the lesion is completely removed, long term review is still usually indicated since leukoplakia can recur, especially if predisposing factors such as smoking are not stopped.

Medications

Many different topical and systemic medications have been studied, including anti-inflammatories, antimycotics (target Candida species), carotenoids (precursors to vitamin A, e.g. beta carotene), retinoids (drugs similar to vitamin A), and cytotoxics, but none have evidence that they prevent malignant transformation in an area of leukoplakia. Vitamins C and E have also been studied with regards a therapy for leukoplakia. Some of this research is carried out based upon the hypothesis that antioxidant nutrients, vitamins and cell growth suppressor proteins are antagonistic to oncogenesis. High doses of retinoids may cause toxic effects. Other treatments that have been studied include photodynamic therapy.

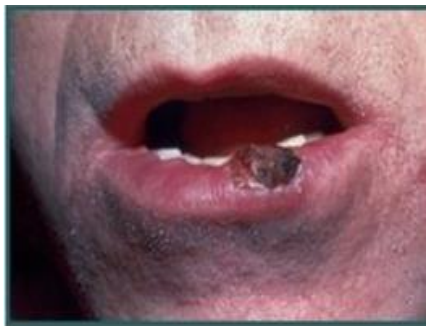
Cornu cutaneum

Cornu cutaneum is limited hyperplasia of the epithelium with strong hyperkeratosis that resembles horn taking into account its appearance and solidity. It occurs on vermilion border, more often on lower lip, and is characteristic for people over 60, it is nontender. Its color is grey or grey-

brown, diameter is up to 1 cm, length is up to 1 cm. Cutaneous horn is a long lasting disease (for years). Emergence of inflammation and induration around horn base, intensification of comification are the main signs of its malignization. The diagnosis is confirmed after removal of lesion and its histological examination. Surgical treatment is removal of cutaneous horn in the limits of healthy cells.

## TOPIC 39.

## OBLIGATORY PRECANCERS OF THE MUCOUS MEMBRANE OF THE ORAL CAVITY AND RED LIP BORDER.



Keratoacanthoma

Keratoacanthoma is epidermal benign tumor that develops quickly and regresses spontaneously. The disease occurs on vermilion border, rarely on tongue. Keratoacanthoma appears as a grey-red solid papule with choanoid deepening in the center filled with horn mass that can be easily removed. The tumor grows rapidly and in a month it reaches its maximum dimensions (2.5 x 1 cm).

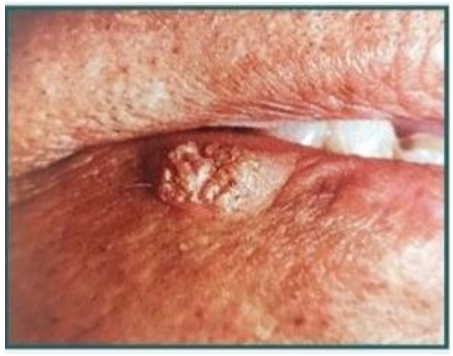
Keratoacanthoma is nontender, movable and do not infiltrate surrounding tissues. In 6-8 months the tumor either regresses spontaneously and disappears leaving a scar or becomes malignant one and causes cancer. Keratoacanthoma must be distinguished from verrucous carcinoma and precancer. Cancer has firmer texture, solid base, after removal of horn masses bleeding emerges. Surgical treatment of keratoacanthoma is ligatory.

Morbus Bowen

Bowen was the first to describe this disease in 1912. From the very beginning it appears to be cancer in situ.

Clinical presentation. The affected area is as a rule single hyperemic bright red spot, smooth or with velutinate surface due to small papillary projections. The central area resembles leukokeratosis with pit-and-mount surface or lichen acuminatus with comification focuses on hyperemic background. Due to atrophy of' mucosa, the focus falls in slightly compare to surrounding areas, slightly bleeding erosions can appear on it. The affected area is from 1-2 mm to 5-6 cm, its contours are not smooth but rather distinct. Induration in its basis is not determined. If lesion is localized on the tongue, lingual papillae at affected area disappear. Regional lymph nodes are not palpable as a rule. Subjective sensations are minor but in cases of erosions tenderness can be present. Clinical presentation of Morbus Bowen on oral mucosa is not always apparent. The disease can manifest only by the small area of hyperemia or resemble leukokeratosis without evident inflammation. The disease may be present for indefinite time, in some cases invasive growth advances and traumatization accelerates this process, in other cases the disease remains at stage of cancer in situ. The diagnosis must be confined by histological study. Pathomorpbologically morbus Bowen is characterized by signs of intraepithelial epidermoid cancer: polymorphism of spicular layer cells up to atypia, increase of mitosis number, its impairment, presence of giant cells, multinuclear cells, acanthosis, and in several cases hyperkeratosis and parakerathosis. The basal membrane and basal layer are preserved. There is small infiltration with lymphocytes and plasmocytes at the upper part of stroma.

Differential diagnostics includes leukoplakia, lichen acuminatus, chronic traumatic affection.



Precancerous verrucous

-Dtvelops in vermilion borders of lover lip

-Hemispherical shape with size of 4-10 mm

-Protrudes elevated above the level of vermilion borders with 4-5 mm

Treatment:

-Surgical excision within the limit of healthy tissue

-Curodestruction

Cheilitis abrasiva praecancrosa Manganotti

This type of precancerous lesions was identified and described by Manganotti in 1933. It occurs predominantly in men over 50 years. Traumas, insolation, herpes may cause the emergence of this disease.

Clinical presentation. Unlike low-grade limited or expanded chronic catarrhal inflammation of the lower lip one or rarely several red anabrosis with flat surface are seen. They can be sometimes covered with tight blood-tinged or serous crust, which is hard to remove. Anabrosis uncovered with scab tends to bleed. Induration at the base of the lesion is absent. Anabrosis can be hardly treated with ointments and applications of topical medications. Existing for a long time they can epithelize but then they appear again at the same or other areas. Histologically the deficiency of epithelium is observed as well as inflammatory infiltration in underlying connective tissue. Epithelium on the anabrosis edges is in the state of acanthosis or is atrophic. Epithelial bundles deviate deep into stroma. In some areas acanthocytes demonstrate different rate of discomplement and atypia. Cytological examination can reveal the signs of dyskariosis of epithelial cells, inflammatory elements but more often only inflammation. The process lasts from 1-2 months up to many years. If not treated it undergo malignization, which manifests clinically by induration of the anabrosis base and surrounded areas, emergence of papillary projections on the surface of anabrosis, its bleeding and comification around the anabrosis. The diagnosis is confirmed by revelation of atypical cells in scrapes from the diseased area or by results of histological examination. The differential diagnostics must be performed with anabrosis forms of leukoplakia, lichen acuminatus, erythema centrifugum, vesicular fever, herpes iris, actinic cheilitis, herpetic anabrosis.

Treatment. It is necessary to remove properly all local irritators, and then oral cavity sanation must be executed including adequate prosthesis, smoking and consumption of irritating food must be forbidden, removal of insolation is also recommended. It is necessary to reveal and to treat accompanying diseases of other organs and systems. Vitamin A is prescribed intraoraly (solution of retinol acetate in oil 3.44% or solution retinol palmitate in oil 5.5 %) 10 drops 2-3 times per day, as well as other polyvitamins. Applications with oil solution of vitamin A are prescribed topically, in case of baseline inflammation — ointments with corticosteroids and antibiotics. Nonsurgical therapy must not be carried out for more than 1 month. The best results are achieved by surgical removal of focus in the limits of healthy tissues. Only in case of Manganotti cheilitis the nonoperative treatment is permitted. The treatment of all types of obligate precancer is surgical procedure — full dissection of the diseased area in the limits of healthy tissues followed by immediate histological examination. Dissected tissue is surveyed by means of preparation of serial sections. Operation must be preceded by oral cavity sanation and removal of irritators. If operative treatment is impossible, radiation therapy is required.

## TOPIC 40.

## DENTAL CHRONIOINTOXICATION. ETIOLOGY. PATHOGENESIS. CLINIC, DIAGNOSIS, TREATMENT AND PREVENTION OF STOMATOGENIC CHRONIOINTOXICATION.

Dental fluorosis occurs as a result of excess fluoride ingestion during tooth formation. Enamel fluorosis and primary dentin fluorosis can only occur when teeth are forming, and therefore fluoride exposure (as it relates to dental fluorosis) occurs during childhood. In the permanent dentition, this would begin with the lower incisors, which complete mineralization at approximately 2-3 years of age, and end after mineralization of the third molars. The white opaque appearance of fluorosed enamel is caused by a hypomineralized enamel subsurface.

With more severe dental fluorosis, pitting and a loss of the enamel surface occurs, leading to secondary staining (appearing as a brown color). Many of the changes caused by fluoride are related to cell/matrix interactions as the teeth are forming. At the early maturation stage, the relative quantity of amelogenin protein is increased in fluorosed enamel in a dose-related manner. This appears to result from a delay in the removal of amelogenins as the enamel matures. In vitro, when fluoride is incorporated into the mineral, more protein binds to the forming mineral, and protein removal by proteinases is delayed.

This suggests that altered protein/mineral interactions are in part responsible for retention of amelogenins and the resultant hypomineralization that occurs in fluorosed enamel. Fluoride also appears to enhance mineral precipitation in forming teeth, resulting in hypermineralized bands of enamel, which are then followed by hypomineralized bands.

Enhanced mineral precipitation with local increases in matrix acidity may affect maturation stage ameloblast modulation, potentially explaining the doserelated decrease in cycles of ameloblast modulation from ruffle-ended to smoothended cells that occur with fluoride exposure in rodents. Specific cellular effects of fluoride have been implicated, but more research is needed to determine which of these changes are relevant to the formation of fluorosed teeth. As further studies are done, we will better understand the mechanisms responsible for dental fluorosis.

## TOPIC 41.

## DIFFERENTIAL DIAGNOSIS OF NON-CARIOUS LESIONS OF HARD TISSUES OF TEETH.

Noncarious lesions, which appear after the eruption of teeth

Abrasion of the hard teeth tissues

Teeth are obliterated not only by the contact of occlusal surfaces of teeth-antagonists, but also in the section of interdental contact points. As a result of elasticity of the collagenic fibers of periodontium teeth insignificantly move along each other, which is called the physiological mobility (amplitude of 0,1-0,2 mm). In this regard, with age as a result of abrasion the planar contact appears between teeth instead of the point one.

Physiological abrasion is observed both in temporary and in permanent occlusion, for example: temporary and permanent incisors erupt with serrations, which obliterate quite rapidly. In temporary teeth the process of abrasion is more expressed and it barely depends on character of food. When biting and chewing food the abrasion and grinding of the occlusal surfaces of dental crowns occur. The smooth polished areas (facets), whose arrangement depends on the form of occlusion, are forming in superficial layer of enamel. The degree of physiological abrasion of teeth has risen with the age.

Pathologic abrasion of teeth is the state of their increased abrasion, when the atypical contact areas, surrounded by the sharp edges of the preserved enamel, are formed in the tooth or teeth in a short time. In this case the teeth lose their anatomical form, interrelation in dentitions changes, occlusion reduces and dentitions cannot carry the functional load without subsequent damage of hard tissues.

Clinical picture of pathologic abrasion.

Complaints:

1.To the increased sensitivity to the temperature stimuli.

2.With the deepening of process pain from chemical stimuli can be joined, and then — from mechanical stimuli.

3.Subsequently, as a result of intensive postponement of replacing dentine, the increased sensitivity can decrease or disappear.

4.In spite of the significant abrasion, in most people the sensitivity of pulp remains or decreases insignificantly.

Objectively:

1.Abrasion manifests as disappearance of the cusps of occlusal surface, cutting edges of frontal teeth visually.

2.Specific grinding in occlusal surfaces and cutting edges of teeth-antagonists occur.

3.Characteristic, smoothly polished, bright surfaces of abrasion (facets), surrounded by protruding sharp edges of enamel are formed. The surfaces of abrasion are different and they depend on the form of occlusion, influence of exogenous and endogenous factors, influence of local mechanical factors.

4.Once the enamel is lost completely, considerably process of abrasion of dentine accelerates, which leads to the formation of sharp edges and changes of the anatomical form of teeth. The hard tissues of dental crowns can obliterate almost to the level of gums in the neglected cases of pathologic abrasion.

5.With generalized abrasion the lower part of face decreases and occlusion reduces sharply, that causes the disease of temporal-mandibular joint, periodontium and reduction of effectiveness of the chewing of food.

6.Surfaces are damaged with caries very rarely, which is explained with the loss of retentional points for the delay of food and microorganisms.

Treatment of pathologic abrasion consists in:

1.Elimination of etiological factors;

2.Elimination of hyperesthesia;

3.Polishing sharp edges of teeth and filling defects;

4.Prosthetic treatment to eliminate separate defects and to increase the height of occlusion with fixed or removable denture.

Wedge-shaped defect

It is localized in precervical section. It has the form of the wedge, whose edge is directed toward the cavity of dental crown.

Clinical picture of wedge-shaped defect :

Complaints:

•Complaints cannot appear at the initial stages of development of the wedge-shaped defect.

•The sensation of pain or the increased sensitivity from acid, sweet, cold and hot appear with the recession, at the last stages — aesthetical defect.

•Complaints have not been ever, because the slow development of process conduce to deposition of tertiary dentine, but pulp gradually atrophies.

•As a rule, complaints disappear with an even bigger recession of defect, in this case the cavity of tooth is obliterated and it looks as dark point.

•The subsequent progress of defect can lead to the fracture of crown. Rarely defect is complicated with caries.

Objectively: at the early stages of its development wedge-shaped defect does not have a form of wedge, but superficial scratches or thin fissures, or slots are revealed. Then these recesses begin to widen and, reaching certain depth, more and more they acquire the form of a wedge. Both walls are smooth, shining, polished, unconverted in colour.

Defects are localized on the vestibular surfaces predominantly and very rare — on lingual (palatine). They can be single, but more often — multiple and they are placed on the symmetrical teeth, they develop very slowly.

Treatment of wedge-shaped defect

The general treatment provides for prescription the microelements and vitamins for the purpose of strengthening of structure of teeth internally and the removal of hyperesthesia.The wedge-shaped defects, whose depth exceeds 2 mm, must be filled (before this course of remineralized therapy is prescribed).In certain cases, in danger of destruction of the tooth crown the tooth is covered with an artificial crown.

Erosion of hard teeth tissues

This is the progressive loss of hard teeth tissues (enamel or dentine) with unexplained etiology. In contrast to the carious lesions, the appearance of erosion is not related to the influence of microorganisms.

Clinical picture. Erosions are localized on the symmetrical surfaces of central and lateral incisors of upper jaw, and also on canines and premolars of both jaws, they does not occur on incisors and molar of the lower jaw. As a rule, there are no single lesions, usually two and more symmetrically placed teeth are involved.

Since the beginning of development erosion looks as defect of oval form, which is placed in the transverse direction on the most convex part of vestibular surface of crown. The bottom of erosion is smooth, shining, solid. A constant recession and the expansion of the limits of erosion lead to the loss of entire enamel on the vestibular surface of tooth and part of the dentine.

Subjectively. Pains appear rarely or expressed weakly. This is connected with the slow development of process, as a result of which occurs the postponement of replacing dentine. The pains can appear from all forms of stimuli — temperature, chemical and mechanical with increasing depth of lesion. The periods of appearance of pain (active phase) alternate with their absence (stopped erosion).

Treatment of the erosions of hard tissues. It is necessary to take into account the activity of process and character of the associated somatic disease (which is treated by a specialist).

Necrosis of hard teeth tissues

This is unique noncarious lesion, which is developed under the influence of some unmicrobal external factors (acids, ionizing emission), it is characterized with the progressive and irreversible destruction of hard teeth tissues and can to lead to total loss of teeth.

Necrosis is caused by both exogenous and endogenous (disturbance of the activity of endocrine glands, diseases of central nervous system, chronic intoxication of organism) factors. Types of necrosis are: precervical and chemical necrosis.

Acidic (chemical) necrosis appears in people, who work in the manufacturing mostly of inorganic acids, which evaporate and fall into the saliva. In this case the saliva acquires acid reaction and decalcifies the hard teeth tissues. This lesion is possible, but in the mild form, when using 10 % of hydrochloric acid with achylic gastritis. Oral respiration favours to the appearance of acidic necrosis.

At acidic necrosis, at first, frontal teeth are affected, enamel begins to disappear from the cutting edge, and then process passes to the vestibular surface. At first, teeth become matted, then — dull gray, sometimes yellow or brown. The dental crowns become shorter; the cutting edge acquires oval form. Gradually the crowns of front teeth become destroyed to gingival edge, and premolars and molars erase badly.

Typical complaints in patients appear already at the initial stages: the sensations of numbness and soreness in the teeth, the sensation of adhesion of teeth with occlusal contact appear.

The phenomena of hyperesthesia can appear. If replacing dentine manages to form, then painful sensation subsides.

Treatment consists in:

•Elimination of the reason for the acidic necrosis (with hydrochloric acid must drink through the straw);

•Remineralizing therapy with the preparations of calcium and fluorine;

•Alkaline gargles.

Hyperesthesia of hard teeth tissues

Hyperestesia is the increased painful sensitivity of the hard teeth tissues in response to the action even of insignificant temperature, chemical and mechanical stimuli, which rapidly disappears. Usually these phenomena are constant, but sometimes temporary calming or curtailment of pain (remission) can observe. Sometimes pain can radiate to the adjacent teeth. More often this phenomenon is observed with the pathology of the hard tooth tissues of noncarious origin, and also with the caries and the diseases of periodontium.

Hyperesthesia can be:

• local (it appears from the action of local stimuli);

• systemic or generalized, which appears with neural and mental diseases (psychoneuroses), endocrinopathies, metabolic disturbances, diseases of digestive canal, climax, infectious diseases.

The most important is local treatment of hyperesthesia. These groups of medicines can be used for treatment:

I.Cauter medicines.

II.Medicines with dehydrating action.

III.Medicines with biological action.

IV.Anesthetics and analgesics.

V.Adhesive systems of light-cured composite materials.

Clinical efficiency and complications for home methods of tooth bleaching, dental treatments and preventive measures.

Differential diagnostics of fluorosis is made with enamel hypoplasia, spotted form of enamel hypoplasia is the most difficult to differentiate.
In case of systemic hypoplasia, as well as in case of fluorosis, deciduous teeth are rarely affected. Chalk-like stains in cases of both fluorosis and hypoplasia, locate symmetrically on teeth in areas of dental crown that are atypical for caries - labial and lingual surfaces, on cusps and incisal edges. In case of fluorosis the stains are pearl-white, glossy, painless under the probe; they gradually transform into unchanged enamel. In case of hypopla- sia stains are white, dense, and glossy; they have accurate limits. In case of fluorosis chalk-like stains fluoresce light blue; the same does the intact dental enamel. In case of enamel hypoplasia stains fluoresce light yellow. In case of fluorosis pigmented stains fluoresce red-brown. Fluorosis stains are permanent, they do not change; caries never develops in these areas. In case of enamel hypoplasia the spots are often complicated with caries.

# TOPIC 42.
# MODERN METHODS OF TEETH WHITENING AND RESTORATION WITH NON-CARIOUS LESIONS.

Hypoplasia of the hard tissues of the tooth Timely medical care for hypoplasia is of great not only aesthetic but also psychological importance, as it helps to eliminate unwanted emotional stress. Local treatment primarily begins with professional oral hygiene; remineralizing therapy – Ca, P-containing drugs (10-20 procedures daily or every other day), fluorine-containing drugs (topically); In addition to these measures, if necessary, the patient should be trained in individual oral hygiene, and the selection of hygiene products (pastes, toothbrushes). Treatment of local hypoplasia is carried out using remineralizing and bleaching agents, and in case of significant destruction of enamel, restoration of the tooth crown with self-or light-hardening composite materials. General treatment includes the appointment of calcium preparations, trace elements, vitamins (course for 30 days, twice a year); as well as a balanced diet with limited intake of refined carbohydrates. If necessary, the defects are filled with composite and glass-ionomer cements. With a strongly thinned cutting edge and aplasia of the enamel on the tubercles of premolars and molars, the covering of such teeth with artificial crowns is indicated. The criteria for the effectiveness of treatment is the restoration of the anatomical shape of the tooth.

Enamel hyperplasia often does not require treatment. With significant changes in the shape of the teeth, grinding of the enamel is carried out with a certain correction of the shape of the tooth, self- or light-curing with composite materials.
Tooth fluorosis Treatment of dental fluorosis is carried out in a comprehensive manner, taking into account the severity of the disease, the general condition of the body and the influence of endemic factors:

• termination or at least restriction of access to the patient's body of increased concentrations of fluoride with drinking water and food; • weakening the toxic effect of high concentrations of fluoride on the body as a whole and on dental tissues in particular by prescribing milk and dairy products, calcium preparations for a month. Local treatment of dental fluorosis should be carried out differentially, taking into account the severity of the disease and the reactivity of the body. For all manifestations of fluorosis, patients are prescribed 2-time brushing of teeth with pastes containing calcium glycerophosphate (toothpaste "Pearl", "Arbat") or remodent (toothpaste "Remodent"). If necessary, sanitize the oral cavity and treat periodontal diseases (gingivitis, periodontitis). For the I degree of severity of fluorosis, local treatment, as a rule, is limited to this. Brown pigmentation is a constant sign of more pronounced dental fluorosis.

Wedge-shaped defect To reduce mechanical stress, soft toothbrushes, non-abrasive toothpastes are recommended. The movement of the toothbrush while brushing your teeth should be vertical and circular. Effective is temporary filling with cement and composite materials with the addition of remineralizing agents in their composition (before mixing) in a concentration of up to 5% (sodium fluoride). In the presence of severe defects in hard tissues, filling is recommended. The most convenient are glass-ionomer cements, compomers, and composite filling materials, which close teeth defects without preparation or preparation after conditioning and applying adhesive. With deep wedge-shaped defects, the manufacture of artificial crowns is necessary. Patients are recommended treatment of concomitant pathology, they are prescribed Ca preparations with microelements (calcium-D3 Nikomed, etc.). They recommend paying attention to diet (limiting the use of easily digestible carbohydrates, etc.). The criteria for the effectiveness of treatment is the stabilization of the 7 pathological process in the form of restoration of the anatomical shape, function and cosmetic qualities of the tooth.

Erosion of hard tooth tissues In case of erosion of hard tooth tissues, treatment should be carried out taking into account the effect of the etiological factor, the depth of the pathological

process, the presence of a concomitant somatic disease. To stabilize the pathological process, applications or electrophoresis of remineralizing agents are used: prescribed daily or every other day. Use temporary filling of cement lesion sites with remineralizing agents. If necessary, use modern light-curing filling materials for the restoration of defects in hard tooth tissues. If necessary, the patient is trained in individual oral hygiene, the selection of hygiene products. Hypersensitivity

To eliminate hyperesthesia, pastes were widely used, which include alkali: sodium bicarbonate, sodium, potassium, magnesium carbonates, as well as substances that can rebuild the structure of tooth hard tissues: sodium fluoride, strontium chloride, calcium preparations, etc. According to modern concepts, fluorine ion is able to replace the hydroxyl group in hydroxyapatite, turning it into a more stable compound - fluorapatite. A good effect of the treatment of hyperesthesia is observed from the use of remineralizing pastes "Pearls", "New Pearls" or "Sensodyne" and "Sensodyne F". Currently, with hyperesthesia of tooth tissues, remineralizing therapy is widely used. The lesion areas are coated with fluorinated varnishes, light-cured bonds (4th generation of adhesives) or enamel-dentin adhesives. In recent years, to prevent hyperesthesia, special preventive systems based on composites (the so-called desensitizers) are used for this purpose, for example: Seal & Protect from Dentsply, Super Seal from Bisco and others. Effective drugs from modern drugs for eliminating hyperesthesia are drugs for deep fluoridation according to the Knappvost method ("Dentin Fluid", "Enamel Fluid"), "Gluftored", VladMiva.

## TOPIC 43.

## PHYSICAL FACTORS IN THE DIAGNOSIS AND TREATMENT OF NON-CARIOUS DENTAL LESIONS.

Hypoplasia of dental hard tissues

In the local treatment of hypoplasia of dental hard tissues, after professional oral hygiene, physiotherapeutic treatment is often prescribed, namely: electrophoresis of a 10% solution of calcium gluconate or calcium chloride, 2.5% calcium glycerophosphate solution (5-6 sessions according to Yu.A. Fedorov) electrophoresis of 1-2% sodium fluoride solution on tooth enamel, is used immediately after application of Ca, P-containing preparations (3-4 sessions according to P.A. Leus).

The classification of dental fluorosis is based on the determination of the ohmic resistance of hard dental tissues and the fluorescence of enamel in ultraviolet rays and the clinical manifestations of the disease. Nikolishina (1999). With mild degrees of fluorosis, the electroconductivity of hard tissues 5 does not change and the enamel shows a bright light blue fluorescence. A decrease in the electrical resistance of hard dental tissues with quenching of the primary fluorescence of the enamel indicates severe manifestations of dental fluorosis and the presence of calcium fluoride compounds on the enamel surface. Consequently, the more the ohmic resistance of the hard tissues of the teeth decreases and the more pronounced the quenching of the primary fluorescence of the enamel, the more severe the manifestations of the disease, that is, these objective indicators reflect the severity of the disease. According to the classification of A.K. Nikolishin, based on the determination of fluorescence in UV rays and indicators of ohmic resistance, three degrees of severity of fluorosis are distinguished: mild, moderate and severe. To determine the severity of dental fluorosis, it is first necessary to determine the ohmic electrical resistance of dental tissues and the fluorescence of the affected enamel in UV rays and light primary fluorescence. For the differential diagnosis of fluorosis of teeth with hypoplasia, in particular with a spotted form of hypoplasia, a fluorescent method is used. In UV rays, chalk stains with fluorosis fluoresce with a light blue glow and do not differ from the fluorescence of intact teeth. In case of hypoplasia, the spots fluoresce with a light yellow glow. Areas of pigmentation, regardless of their intensity in fluorosis, fluoresce with a red-brown glow. In hypoplasia, enamel pigmentation can be traced in depressions, furrows, in places where plaque accumulates, it is often poorly expressed and therefore does not fluoresce. With pronounced forms of fluorosis without brown color of the teeth, either fragmentary or complete quenching of primary fluorescence appears. In furious and destructive forms of hypoplasia, the fluorescence of the affected areas is light yellow, as in the spotted form of hypoplasia; quenching of primary enamel fluorescence is not observed. Fluorosis spots are relatively constant and stable in their development. In the case of a child living in an area with an optimal or low fluoride content in drinking water, stains may decrease or disappear altogether over time. With hypoplasia, the spots do not disappear with the age of the child, the process progresses and leads to the development of superficial caries in their place. The electrical conductivity of fluorotic spots and spots in systemic hypoplasia is not changed. However, with the so-called "chalk degeneration" of the enamel and with the destructive form of fluorosis, the ohmic resistance of hard dental tissues is significantly reduced due to the formation of calcium fluoride. In furious and destructive forms of hypoplasia, the electrical conductivity of the hard tissues of the tooth changes due to a decrease in the thickness of the enamel. Treatment. To enhance the whitening effect, one of the physical factors is additionally used - electrophoresis of 5% calcium chloride No. 10 according to the method of Nikolishin A.K., heat, UV irradiation, laser beams (Nikolishin 6 A.K., Ilenko N.N.), etc.

Wedge-shaped defect With the initial manifestations of a wedge-shaped defect, conservative methods are used to stabilize the pathological process, namely the application or electrophoresis of a 10% calcium gluconate solution 2% sodium fluoride solution 2.5% calcium glycerophosphate solution.

Erosion of hard tooth tissues To stabilize the pathological process, applications or electrophoresis of remineralizing agents are used: 3-4 electrophoresis procedures are prescribed daily or every other day, lasting 10-15 minutes. In the next 3 visits to the erosion site for 2-3 minutes. apply acidified fluoride gel in 0.1 M solution of phosphoric acid or perform temporary filling with cement with remineralizing agents. The treatment is completed by coating the affected surface with fluorine varnish. According to the method of P.A. Leus, for electrophoresis of 10% calcium gluconate solution, you can use the ELOZ-1 apparatus: current strength 30-50 μA, procedure duration 5-10 minutes; after each procedure of electrophoresis for erosion for 2-3 minutes. apply a tampon moistened with 2% sodium fluoride solution. The course of treatment is 10-15 procedures. The number of visits for the application method is 15-20. To remineralize hard tissues, you can use a two-component remineralizing solution consisting of 10% solutions of calcium nitrate and acid ammonium phosphate.

Increased sensitivity Yu.A. Fedorov (1970) to eliminate systemic hyperesthesia recommends taking calcium glycerophosphate at 0.5-1.0 for a month and electrophoresis of a 2.5% solution of calcium glycerophosphate topically in the amount of 10 sessions. P.A. Leus (1978) recommends for the elimination of foci of demineralization in case of caries and weak forms of non-carious lesions (erosion, wedge-shaped defect and others), the application of a 10% solution of calcium gluconate and 2% sodium fluoride solution (5-6 sessions) or the application of calcium gluconate and the introduction of 2% sodium fluoride solution using electrophoresis (Eloz-1 apparatus with a current strength of up to 50 μA).

## TOPIC 44.

## DIFFERENTIAL DIAGNOSTICS OF CARIOUS LESIONS OF HARD TISSUES OF TEETH.
## CLASSIFICATION OF CARIES

Clinical features of caries are enough various: from a chalky white spot on the surface of enamel to an expressed destruction of hard tooth tissues. These numerous forms of caries, per se, are stages of tooth destruction changing each other consistently (if untreated). The progress of caries process leads to destruction of entire thickness of hard tooth tissues, perforation of pulp chamber and development of inflammation of pulp (pulpitis) or periodontal ligament (apical periodontitis). Therefore pulpitis and apical periodontitis, another tissues of maxillo-facial region, which arise because of caries process, are called caries complications.

Depending on the depth of caries lesion of enamel and dentine, caries is divided into superficial, middle and deep. Depending on the caries course, acute (rampant) and chronic caries are distinguished.

Incipient caries (caries incipience). A characteristic feature is development of demineralization on the enamel surface. As a result incipient white spot lesions and stained, roughened, partially remineralized incipient lesions of enamel develop. A carious cavity in enamel is forming.

Superficial caries (caries superficialis) — there is a carious defect in enamel, dentino-enamel junction is not destroyed by caries process.

Middle caries (caries media) — carious cavity in dentin is formed: in mantle dentin. This layer of dentin is juxtaposed with enamel and converted into the initial layer of dentin from the basement membrane.

Deep caries (caries profunda) — carious cavity in mantle dentin is formed: in circumpulpal dentin. This layer of dentin is localized very close to pulp. The carious cavity can divide from the pulp chamber only very thin partition or only the layer of secondary dentin.

Clinical feature of caries

Incipient caries. Patients complain of spots (white, chalky white, opaque or pigmented), rarely of feeling insignificant sensitiveness, sorenesses of the mouth from different irritants, mainly chemical (sour, sweet).

In the acute (rampant) course on the limited areas of dental enamel the opaque, deprived of natural transparency, chalky white spots appear. At first, spots are small, but, gradually increase in size. They are often located on occlusal surfaces in retentive points: pits and fissures of occlusal surfaces of teeth, cervical areas. In children they are often localized on a vestibular surface and cervical areas. For the best revealing of caries spots it is recommended to remove debris from the spot surface and dry up the tooth crown: intact enamel saves its natural transparency and brilliance, while the surface of caries spot loses transparency and becomes opaque. When probing a roughness, insignificant pliability and painfulness of their surface can be revealed.

The histological features at an initial caries are characterized by development of different degrees of enamel demineralization. In enamel section the body of caries lesion has the form of a triangle with the basis directed to enamel surface. In the study in the polarized light depending on the structure changes in enamel lesion a few areas are distinguished. The most demineralized is

subsuperficial lesion layer, which is covered with mineralized superficial enamel layer. This interesting phenomenon is explained by remineralization processes of carious lesion by the mineral components of saliva. If oral fluid is unable to provide remineralization of the demineralized enamel area, rapid development of caries lesion occurs.

Superficial caries. In some period of time in the center of carious spot the superficial layer of enamel loses the integrity and defect appears in enamel. At acute superficial caries patients complains of insignificant pain, more frequent of feeling soreness of the mouth and affected tooth, caused by chemical irritants, which at once disappear after cessation of irritant actions. Sometimes shot-term pain from thermal and mechanical irritants occur, more often in place of localization of carious lesion.

At the examination of tooth in the area of chalky white color a lesion of a shallow enamel defect (cavity) is determined, placed within the enamel borders. The enamel wall with lesion is softened, yellow-grey and a little sensible when probing. Sometimes it can be only rough surface, but after removing the softened enamel surface the lesion (cavity) is found.

The chronic superficial caries course is mainly painless. Insignificant pain from chemical irritants rarely occurs. But it disappears immediately after cessation of irritant action. A small enamel lesion (cavity) with enough dense yellow-brown or brown enamel walls is revealed on the enamel surface. The cavity has a wide, exposed inlet without overhanging margins. Probing of carious defect is practically painless. When superficial caries is localized in fissures the margins of lesions remain undamaged.

Superficial caries is diagnosed on such grounds: a) patient complaints about short-term painful feeling mainly from chemical irritants; pain disappears after cessation of irritant action; b) revealing of shallow carious cavity, located within enamel borders, or of fissures pigmentation on occlusal surface, in which the softened demineralized enamel is revealed when probing; c) painful preparation of hard tooth tissues especially at the dentinoenamel junction.

Middle caries (caries media). After destruction during the pathological process of dentinoenamel junction caries quickly begins to spread to the dentine. Middle caries (caries media) is pathological condition, when a carious cavity is located in mantle dentin. Patients with acute middle caries often complain about painful feeling. More often the pain has weak intensity and appears only under the action of irritants: chemical, thermal, mechanical. On the tooth surface there is a chalky white carious spot with the enamel defect in a center. Examination of the cavity is difficult because of its narrow inlet. The cavity usually has a depth of 1,5-2 mm, it is filled with food debris and softened dentin. Complete examination of carious cavity is possible only after removing of overhanging chalky white enamel margins with special instruments (burs, excavators). The cavity is wider near the dentinoenamel junction and it is narrowing towards the pulp gradually. The softened dentine which covers the cavity, is grey-white or yellow, rarely — pigmented. The degree of dentine softening depends on the activity of caries process: at acute (rampant) caries the hard tooth tissues are more softened like a cartilage, at chronic course it can be harder and pigmented. Probing of carious cavity is practically painless except the dentinoenamel junction.

Chronic middle caries has practically little clinical symptoms. In some cases weak pain can occur because of action of chemical, rarely thermal and mechanical irritants and it disappears at once after their cessation. When examining the carious cavity a rather wide inlet is revealed, it is located in mantle dentin, the depth of cavity is 1,5-2 mm depending on the surface of the tooth. The carious cavity is covered with rather dense pigmented dentin, floor and walls of the cavity are painless when probing. During electric pulp testing (electroodontodiagnostic method) the pulp reacts on the current strength of 6-12mcA.

Deep caries (caries profunda). It is characterized by formation of carious cavity which affected almost all layers of dentine practically to pulp and it is located in circumpulpal dentin. Patients with acute deep caries complain about causal pain which appears because of action of thermal, mechanical, chemical irritants and disappears after their cessation. Inserting into the carious cavity of cotton tampon with hot (no more than 50° C) or cold water, and also ether is usually accompanied by sharp pain reaction, however, pain disappears after the removal of irritants from the cavity. Carious cavity is located within the limits of circumpulpal dentin with the overhanging margins of enamel. Enamel around the inlet has softened chalky white color. The carious cavity is filled with grey-whitish or yellow softened dentin. When probing a painful area on the cavity floor and dentinoenamel junction is revealed. Often it is the places of projection of pulp horns, which directly react on irritants; however, perforation of carious cavity is not observed. At acute deep caries, probing of caries cavity floor must be made very carefully. At the points of pulp horns projection the dentinal wall is very thin, the dentine is softened and can be easily pierced with a probe and injure pulp. It is accompanied by sharp pain and a blood drop appears in the carious cavity.

At the chronic deep caries there can be no complaints of pain, while insignificant, brief pain after thermal, chemical and mechanical irritants can be revealed. Defect of hard tooth tissues is located in limits of circumpulpal dentin, rather large in size, occupies considerable part of tooth crown. Cavity is wide opened (the overhanging edges of enamel are broken off because of their fragility). As a result, the transversal sizes of cavity exceed its depth. Walls and floor of carious cavity are filled with rather dense, pigmented dentin, but without sclerotic brilliance. Pigmentation of its walls and floor has enough wide spectrum — from yellow-brown to brown and even almost black color. Probing of walls and floor of the cavity is painless, because of development of well expressed areas of transparent and secondary dentin under them. Surface of the carious dentin is rough when probing. The development of such cavity continues for years.

Differential diagnostics of acute initial caries should be held with non - carious lesions — spotted form of system enamel hypoplasia and mild form of fluorosis. A method of vital coloring by 2% water solution of methylene blue is used for differential diagnostics of acute initial caries with non-carious lesions of dental hard tissues. The doctor cleans the enamel surface, washes it with hydrogen peroxide and dries it. Then he isolates the teeth from saliva, applies a coloring solution for 2—3 minutes; washes off the solution with a water. Area of enamel with acute initial caries, unlike hypoplasia or fluorosis, gets light blue color. Coloring level is estimated with 10-point scale of the blue color. 0.1% water solution of methyl red can also be used as a coloring agent. At present time, «Diagnodent» (KaVo, Germany) is the best device for diagnostics of initial caries in fissures and other hard-to-reach dental surfaces. The methodic of use of the «Diagnodent» is presented below: — soft denial deposits are removed from the tooth surface (the deposits may distort dest data); — the surface of tooth is dried; — the examined area is illuminated with a detector, and in a few seconds a digital display shows the results of the survey in a form of digital indices. According to A. Lussi (1995) digital indices 0-14 correspond to normal enamel structure, 15-20 - to caries process within enamel, 21-90 - to caries process within dentine. With the help of «Diagnodent» caries can be detected with 90%-accuracy. Chronic initial caries is characterized by full absence of pain sensations and by formation of pigmented spots (dark brown or black). After drying the spots are matte, their probing is painless, the surface is rough. Its density doesn't differ from that of surrounding healthy enamel. Pigmented carious stains (macula cariosa) are detected in children more rarely than the chalk-like ones. More often they are detected on medial-approximal surfaces of the first permanent molars at the time when these areas become open after the second deciduous molars falling of. Superficial caries in permanent teeth in children replaces white or pigmented spots as a result of progression of destructive changes in enamel. Acute superficial caries is

characterized by softening of affected enamel which can be removed by excavator with slight effort. Most children have no complaints at this stage of the pathological process. Some of them may foci short-term pain (soreness of the mouth) from chemical irritants — sore, sweet, salt; pain is fast relieved after removal of the irritant. Clinically acute superficial caries looks like an area of destruction of enamel with typical localization. Defects of relief, very rough surface, and stiffness on probing are detected; the carious cavity is located within the enamel layer. Differential diagnostics of acute superficial caries should be made with acute initial caries, acute moderate caries, spotted form of enamel system (or local) hypoplasia, or erosive form of fluorosis. Acute superficial caries differs from acute initial caries by complete destruction of enamel surface and penetration of the pathological process into the enamel. Unlike acute moderate caries, acute superficial caries is characterized by preserved integrity of enamel-dentine junction. In case of acute moderate caries the enamel-den- tine junction is always destructed; the caries process affects dentine; probing of the enamel-dentine junction is painful in case of acute process. Spotted form of enamel system hypoplasia differs from acute superficial caries by multiplicity of defects and their symmetrical location. Hyperesthesia is absent. Hypoplastic defect of enamel is characterized by regular spherical contours with smooth edges. The bottom of the defect is always smooth, solid and shining. Erosive form of endemic fluorosis, as well as acute superficial caries, is characterized by defects within the enamel. But in case of fluorosis defects of enamel can locate at any part of dental surface, including resistant to caries areas. Caries does not develop in these areas. Erosive form ofendemic fluorosis is a result of consumption of water with high content of fluorides (3mg per liter and more); thus, fluorosis can be detected in most children living in these regions. Chronic superficial caries does not cause painful sensations. In case of localization of the carious cavity on the approximal surface, children may complain about food stuck between teeth and inflammation of a gingival papilla — edema, hyperemia, bleeding at touching. Examination of the enamel surface detects a shallow (within the enamel) cavity covered with dense brown or black enamel. The cavity has a wide entrance; probing of the carious defect is painless. In case of fissure location of chronic superficial caries, fissure edges may be preserved. Moderate caries is the most often diagnosed form of caries of permanent teeth in children. The enamel-dentine junction is always destructed. Carious cavity is localized in mantle dentine, thus there is a quite thick layer of unchanged dentine over the pulp chamber. Most children with this form of caries of permanent teeth have no complaints; thus, the lesions are detected during stomatological examination or sanation of the oral cavity by the dentist. In case of acute moderate caries the carious cavity has a narrow entrance which does not correspond to the depth of a dentine lesion. In case of localization of a carious cavity on occlusal surface, only the probe can enter it and then gets stuck there. The enamel edges, covering the entrance to the carious cavity, are chalk-like changed. Therefore, the depth of the dentine lesion, and thus the depth of carious lesion, can only be determined after the preparation of the carious cavity. Carious cavity is filled with softened dentine; it can have unchanged color or yellowish coloring. The level of dentine softening depends on the activity of a carious process. Dentine can have chondroid consistence and can be easily removed with the excavator in permanent teeth with unformed root. Probing of the walls of the carious cavity is painful at enamel-dentine junction; all other areas are painless. Thermal irritantion does not cause any painful sensations, as a rule. Chronic moderate caries is often diagnosed in teeth with formed roots in children of late school age. Clinically there are no complaints, or painful reaction to chemical irritants. Carious cavity is localized in mantle dentine, it has a depth of 1,5—2,0 mm. As a result of slow carious process dentine of walls and bottom is dense and pigmented. Probing of the bottom and walls of a carious cavity is painless. As a Rile, compensate form of activity of carious process is diagnosed in these children. Differential diagnostics. Moderate caries of permanent teeth in children should be differentiated with deep caries and chronic periodontitis. For differential diagnostics of acute moderate caries and acute deep caries it is necessary to assess the depth of carious cavity after

preparation. Acute deep caries is characterized with expressed tooth sensitivity to termal and mechanic irritants. Cold thermal irritants cause painful sensation in the tooth with acute moderate caries; the pain disappears after removal of the irritant. In case of acute deep caries probing of the thinned bottom of the carious cavity is painful due to close location of the pulp; in case of acute moderate caries the walls of the carious cavity (enamel-dentine junction) are more sensitive to probing. Chronic moderate caries of permanent teeth in children should be differentiated from chronic deep caries and chronic periodontitis. Chronic periodontitis in children is characterized by its development with the closed pulp chamber. The color of the tooth and the reaction of the patient to preparation of the carious cavity (if it is performed without anesthesia) are taken into consideration for the differentiation. In case of chronic periodontitis the color of the permanent tooth is changed, the tooth does not react to thermal irritants. The preparation of the walls of the carious cavity in the enamel-dentine junction causes no pain. Gingival mucosa in the projection of the root apex of tooth with chronic periodontitis is characterized by congestive hyperemia; symptom of vasoparesis by Lukomsky is positive. These features make the basis for radiological examination of the affected tooth which helps to make the final diagnosis. Radiological examination is used for assessment of topographic interrelations between the carious defect and the pulp chamber. It allows assess the real depth of the carious cavity, check the presence of complications of caries and make the differential diagnostics. Deep caries is characterized by formation of the carious cavity located within the parapulpal dentine which is thinned and softened. This form of caries is often revealed in permanent teeth in children. Children complain about pain in case of thermal and mechanical irritations; pain disappears soon after the removal of the irritant. During examination the doctor detects the deep carious cavity with a small entrance and overhanging enamel edges. Enamel around the entrance is crisp and chalk-like. Carious cavity is covered with softened dentine of practically unchanged color. Probing cause painful sensations at the area of enamel-dentine junction, as well as at bottom of the cavity and at areas of the thinnest dentine over the pulp (projection of the pulp horns). The carious cavity is not connected with the pulp chamber. Chronic deep caries is often detected in permanent teeth with mature roots in children of older school age. In case of this form of caries there are no complaints about pain, or some painful sensation appear in case of thermal and mechanic irritations. The defect is located within the parapulpal dentine and takes significant part of the dental crown. The entrance of the carious cavity is wide; its transverse size may exceed its depth. The walls and the bottom of the carious cavity are covered with dense, always pigmented dentine. Probing of the walls and the bottom of the carious cavity is painless, as there are zones of transparent and reparative dentine. The surface of the carious dentine is rough and it’s almost impossible to remove it with the excavator. Differential diagnosis. Chronic deep caries in permanent teeth should be differentiated from chronic fibrous pulpitis, pulp hyperemia, acute localized pulpitis or chronic periodontitis. The main difference between the deep caries and acute forms of pulpitis lies in the absence of spontaneous pain in the first case. So, during the questioning the doctor asks about spontaneous pain in the affected tooth in the anamnesis. Chronic fibrous pulpitis is characterized by the longterm pain (up to an hour) caused by thermal or mechanical irritants. Therefore, for the differential diagnosis the doctor pays attention to duration of pain after removal of the irritant. If the pain does not disappear immediately and lasts for some time pulpitis is suspected. After the preparation of the carious cavity its bottom should be carefully examined. Softened dentine of the bottom of the carious cavity in the projection of the pulp horn, and acute pain caused by probing characterize chronic fibrous pulpitis. Electroodontodiagnostics (EOD) is used for differential diagnostics of acute deep caries in permanent teeth with mature roots. Normal pulp electric test data does not exceed 2—6 /

## TOPIC 45.

## MODERN METHODS OF TREATMENT AND PREVENTION OF DENTAL CARIES.

Medical treatment of dental caries

Medical treatment of caries consists of a number of measures of general and local character depending on the stage of development of pathological process and character of its course. On early stages (caries incipience) this complex of measures is directed on the removal or reduction of effect of demineralized factors action, and also on renewal (remineralization) of partly demineralized hard tooth tissues. When the pathological process spreads to enamelodentinal junction, a dentine strikes and a carious cavity appears, conservative (remineralization) therapy can not result in success. It is connected with the fact that hard tooth tissues do not possess property to regenerate the primary form in the area of carious lesion. Therefore for local medical treatment of carious cavities their preparation is used, with the subsequent filling of cavity and renewal of anatomic form of tooth by filling material.

None of varieties of restoration treatment of dental caries can be complete “curing”. Destroyed with caries the hard tooth tissues (and adjoining areas of healthy enamel) are not substituted for newly formed enamel and dentin. Besides, there is no restoration material capable to protect hard tooth tissues from further destructive caries processes during all life. Tooth filling is only symptomatic treatment which does not eliminate the etiologic factors of dental caries.

Therefore the prevention of carious lesions development (prophylactic measures) is the basic principle of carious treatment, than - necessary medical treatment (remineralization therapy) and, in the last turn, forced measure — filling the carious cavity with filling materials, conducted along with the measures of the secondary caries prophylaxis.

Thus, now there are two main methods of local caries treatment: 1) caries treatment without preparation and filling — remineralization therapy, and 2) operative caries treatment by the operative preparation of demineralized hard tooth tissues with the subsequent filling of carious cavity. The choice of treatment method depends on the stage of caries development, activity of caries (acute or chronic), localization of carious cavity, age and general condition of patient.

Conservative medical treatment (remineralization therapy) of dental caries can be conducted only on the stage of absence of a cavity in hard tooth tissues, which is at caries incipience, when anatomic integrity of enamel is not broken.

In general for local remineralization therapy of incipient caries such groups of medications can be used:

1.Means, which influence on mineralization of enamel (they restore and complement ions which are absent in the crystals of enamel at caries; influence on kinetics of mineralization, ect).

2.Means, which prevent adsorption of organic matters (acids, toxins and other products of vital functions of microorganisms) on the surface of hard tooth tissues (desorbents, hydrophobic pellicle coverages, sealants).

Various preparations of fluoride, calcium, phosphor-calcium combination, complexes of mineral components (remodent), etc, are referred to the first group. Their introduction into the demineralized enamel areas renders assistance in remineralization, renewal of mineralization

degree, increases stability of enamel to action of acids and other cariogenic factors. Preparations of fluoride, pectins, natural and synthetic varnishes, and various fissure sealants prevent adsorption of organic matters.

Preparations of calcium and fluoride are often applied for remineralization. The usage of different calcium salts is pathogenic reasonable with its predominance among other mineral elements in the structure of hard tooth tissues apatites (hydroxyapatite, etc.). Efficiency of fluoride preparations application is conditioned with its influence on some mechanisms of pathological process.

There was proposed application and electrophoresis of 1 % solution of sodium fluoride for medical treatment of initial caries. The surface of teeth is carefully cleaned from dental plaques with the help of excavators or special brushes and pastes. Teeth are isolated from saliva and a cotton tampon or a small gauze serviette moistened with 1 % solution of sodium fluoride is put on the carious lesion. Duration of application is 15-20 minutes, during this time the cotton tampon with sodium fluoride solution is been changing 3-4 times. The course of medical treatment consists of 15-20 everyday application. More effective is introduction into enamel of fluoride with electrophoresis with 1 % solution of sodium fluoride that provides more deep penetration of fluoride ions in hard tooth tissues. Duration of procedure is 10-20 minutes, the course of medical electrophoresis treatment is 10 attendances.

Medical forms, which provide adhesion of fluoride preparation to the enamel, in particular, fluoride varnishes and gels, are developed for prolongation of fluoride action on hard, tooth tissues. Fluoride varnish is a composition of natural yellow resins of viscid consistency which contain 1-5 % fluoride (as often as sodium fluoride). For example, widely applied in practice fluoride varnish contains 5 % sodium fluoride, 40 % silver fir balsam, 10 % shellac, 12 % chloroform, 24 % ethyl alcohol, etc. A number of various fluoride varnishes is proposed, for example, “Fluor Protector”, “Duraphat”, “Bifluorid 12” (VOCO), “Belagel F”, “Belak F” (“VladMiVa”, Russia), etc. Teeth are isolated from saliva, dried out and on the area of demineralized enamel the fluoride varnish is applied, which dries up on the enamel surface during 4—5 minutes. Than the patient is recommended not to eat for 2-3 hours, to save this varnish film. It remains on the tooth surface for a few hours, that provides the long contact of fluoride with tooth enamel. Fluoride gels are also applied. In general the course of remineralization therapy consists of 15-20 attendances which are carried out every day or on alternate days.

For local medical treatment of initial caries the preparations of calcium are widely used: 10% solution of calcium gluconate or calcium chloride, 5-10% acidified solution of calcium phosphate, 2,5 % solution of calcium glycerophosphate. They are applied for applications and electrophoresis (the ions of calcium enter from a positive electrode — anode). The course of medical treatment depends on caries activity, number of carious lesions, etc., and it can achieve 15-20 applications or 10-15 procedures of electrophoresis. Efficiency of remineralization action of calcium preparations is multiplied at their combination with phosphorus preparations. As a remineralization preparation the solution, which contains 11 % calcium and 22 % phosphorus is also used (A. Iraig, I. Iraham, 1975). Calcium-phosphate gels, which provide long remineralization action, were also developed. As a remineralization mean fluid containing synthetic hydroxyapatite can be used.

As a result of the effectively carried out medical treatment (remineralization therapy) the demineralized areas of enamel diminish in sizes or disappear. It is possible transition of caries in its stationary form: in such cases a carious spot changes the color from chalky white to yellow or brown and some diminishes in sizes. To determine the effectiveness of the treatment the

remineralization teeth are painted with dyes, for example, with 2 % water solution of methylene blue. If renewal of the degree of mineralization of enamel mineral structures is happened, the area of caries spot is not painted with methylene blue or the degree of staining is insignificant.

It has long been recognized that pits and fissures, especially on occlusal surfaces of teeth, are the most susceptible to caries. At localization of carious spots in fissures of molars and premolars one of the effective methods of medical treatment of such caries is sealing pits and fissures with sealants. Pit-and-fissure sealants provide safe and effective method of prevention of caries. Sealants are the most effective in children when they are applied to the pits and fissures of permanent posterior teeth immediately after eruption of the clinical crowns.

For fissure sealing a number of the most various preparations is used. For filling microspaces which appear in enamel at incipient caries the simple chemical matters can be used: silver nitrate, zinc chloride. Sealing cements (polycarboxylate, silicate, polyacrilic, glass-ionomer cements), composites (chemical and light cured) are actually used for fissures. For greater remineralization action the fluoride preparations are introduced into sealant composition.

At chronic course of incipient caries (yellow and brown carious spots) remineralization therapy is not obligatory. Except for preparation, at chronic caries the pigmented spots can be deleted with the method of enamel microabrasion. Enamel microabrasion is the controlled deleting of enamel, which changed its color, with the mixture of pumice powder and acid, usually hydrochloric acids. For this purpose special device is used — “HANDIBLASTER” (“Bisco”). This method is effective for medical treatment of superficial enamel discolorations (white or brown spots) caused by orthodontic treatment. The device sprays abrasive powder (usually aluminum oxide) on the surface of the tooth for preparation or treatment of the tooth surface before filling.

Operative caries treatment (filling)

During operative caries treatment and filling carious cavity such requirements are recommended to fulfill:

1.Completely remove the hard tooth tissues, destroyed by caries, under sufficient anesthesia;

2.Create the most convenient conditions for firm and reliable fixations of restorations in prepared carious cavity;

3.Unite antiseptic processing with careful drying of prepared hard tooth tissues;

4.Rationally select filling material according to classes of carious cavity and properties of restorative materials, following the rules of its manipulation and inserting into carious cavity;

5.Grinding, finishing and polishing of the restoration.

In caries treatment such steps are distinguished:

•preparation of oral cavity;

•anesthesia:

•preparation of carious cavity (tooth preparation);

•application of isolating or curative liner;

•filling;

•modeling;

•final grinding, shaping and polishing of the restoration.

In general, the objectives of tooth preparation are to: (1) remove all defects andprovide necessary protection to the pulp; (2) extend the restoration (carious cavity); mastication the tooth or the restoration or both will not break and the restoration will not be displaced; and (4) allow for esthetic and functional placement of restorative material.

Dietary control is an important part of caries prevention. Successful dietary advice at the level of an individual (advice that leads to caries reduction) requires effective advice, adequate provision and resources to provide advice, and patient compliance. However, efforts at this level need to be reinforced with population-based strategies for caries prevention.

FISSURE PRESSURIZING

This method is used to pressurize fissures in molars and premolars and natural pits, in teeth of children. It has generally been considered that pit and fissure surfaces of molar teeth usually become carious within 3 years of eruption because enamel is not mineralized yet in fissures (for kids of 6 -8 years old, whose fissures in molars are not enough mineralized). Sealants protect the occlusal surfaces of molar teeth, providing a smooth surface as the morphology of this surface makes it more susceptible to dental caries and favouring plaque stagnation.

Types of materials:

►Acid-etch resin composite sealants (the same stages of applying as light-curing composites – etching, washing, drying stage and sealant placement with subsequent light-curing)

►Glass ionomer cements ( is used when it is difficult to achieve adequate moisture control, as this material is less sensitive to the presence of moisture; is easy to use on children, bonds well to enamel and releases fluoride, providing a potentially cariostatic effect)

Methods:

Non-invasive (for not affected fissures in molars and premolars in teeth that are 2-3 years after eruption; age of children 6-8);

Non-invasive method of fissure pressurizing envisages applying of sealants to the fissures of tooth, that is only cleaned with brush and prophylaxis paste (without fissure preparation by burs) after tooth cleaning follows stage of sealant placement. (see stages of pressurizing)

Invasive (for deep, pigmented fissures, but not affected by caries process)

Invasive method of fissure pressurizing

The same stages are held as in the non-invasive method, but after the tooth preparation, the stage of fissure disclosure is performed with small diamond bur for high-speed handpiece. After the fissure was disclosed, it should be available for good visual examination and for next fissure pressurizing. Invasive method of fissure pressurizing differs from conventional carious cavity preparation by sparing teeth tissues preparation, especially enamel.

Stages of pressurizing:

Tooth preparation

The tooth to be fissure sealed must be plaque free and free from debris. This can be achieved using a prophylaxis cup or brush, with or without pumice or any non-fluoride, non-oil based paste, or by the use of air abrasion. Afterwards, the probe should be run through the fissure system to free

any retained pumice. Following this, the tooth should be thoroughly washed and dried with a three-in-one syringe, and re-examined.

Tooth isolation

Good isolation to keep the teeth dry is critical to successful sealant restoration. A rubber dam to isolate the teeth will provide ideal isolation and will control movements of the tongue but is not always well tolerated by children.

An alternative is to use cotton wool rolls and dry guards; dry guards can be placed over the parotid ducts. Saliva ejectors and high-speed suction are also useful when used in conjunction with other isolation materials.

Moisture-free surface

The tooth needs to be completely dry prior to placement of the acid etchant. The presence of saliva or water compromises sealant bonding and subsequent retention.

Ensure that the three-in-one syringe tip is not contaminated with water coming out of the air tip when drying the tooth by testing on a dry tissue. The inability to obtain good isolation is a contraindication to providing fissure sealants.

Enamel etch

Phosphoric acid etchant gel is applied for the manufacturer's recommended time, usually 30 seconds, to the occlusal surface, ensuring that all buccal and palatal fissures to be sealed are covered. The etching gel is available in syringes with a disposable cannula tip, which allows for accurate placing of the gel. Thirty-seven per cent phosphoric acid is normally used to create microporosities within the enamel, which permits a sealant to flow in and penetrate the etched surface, thereby producing a mechanical lock of resin tags.

Rinsing and drying the teeth

The tooth is fully washed for at least 20 seconds and dried for a minimum of 15 seconds, replacing wet isolation materials with dry ones. The tooth surface should be frosty white, and not shiny. If the tooth becomes wet by contamination from saliva, the etching procedure must be repeated.

Application of sealant

Sealant material is applied, allowing it to flow into the pits and fissures; this reduces the risk of air bubbles forming. Care should be taken to ensure that the pits and fissures are not overfilled. Occasionally the sealant material flows towards the distal surface of the tooth and a probe should be used to ensure that excess material is not left on this aspect as it could interfere with the occlusion or create an overhang, providing a susceptible site for dental caries to occur in the future.

Curing

The sealant should be cured for the manufacturer's recommended curing time, usually between 20 and 30 seconds. A blue light source is used to cure the sealant material. A light-curing shield should also be used to protect the operator's eyes.

Completion

All isolation materials should be removed and the tooth rinsed well. The surface of the sealant should be examined. A probe is used gently to ensure that all fissures are sealed and the

tooth surface is smooth. A successful sealant feels hard and smooth without the presence of bubbles. The occlusion should be checked with articulating paper and any ‘high spots’ should be removed with a finishing bur or white stone as they will interfere with normal occlusion.

Review

The condition of sealants should be reviewed when patients attend for routine dental examinations. This step is crucial to the success and longevity of the material. The sealant should be checked for any loss of the material and also for any discolouration around the edges or underneath the sealant, which may be an indication of dental caries or partial debonding.

Fissure sealants are an effective preventive measure but should be used in conjunction with other preventive regimes, e.g. a low-cariogenic diet and fluoride therapy.

## TOPIC 46.

## PHYSICAL FACTORS IN THE DIAGNOSIS AND TREATMENT OF DENTAL CARIES.

Electroodontodiagnostics (EOD) is used for differential diagnostics of acute deep caries in permanent teeth with mature roots. Normal pulp electric test data does not exceed 2—6 /

ART-technic method of caries treatment in deciduous teeth (Atraumatic restorative treatment) supposes the manual preparation (necretomy) of a carious cavity by excavators of various sizes followed by filling with glass- ionomer cements (GIC). ART assumes following technique: a carious cavity is cleaned of softened dentine with the excavator, dried and filled with glass-ionomer cement. Treatment of initial stages of caries allows complete stopping of its further progress. Atraumatic restorative treatment is accompanied with no (or very low) painful sensations and it does not cause much psycho-emotional sires in little patients. The method can be used at any stage of deciduous teeth's formation in children with nervous hyper excitability.

Chemico- mechanical removal of carious dentine is its chemical softening followed by its careful excavation by manual instruments. «Carisolv» system for chemico-mechanical removal of carious dentine has been created in 1998 in Switzerland. It contains a set of syringes with special gel and special instruments for manual removal of carious dentine from the carious cavity. Treatment technique: 1. Apply the necessary amount of gel on the glass slide immediately before the preparation. 2. Fill the entire carious cavity with gel. 3. In 30 sec begin removing of carious dentine from the carious cavity walls by an instrument of a corresponding form and size from the «Carisolv» set of instruments. 4. The gel is applied to the cavity several times, until all the carious dentine is removed, which is indicated by a pink colored gel and its transparency. Preparation ot the carious cavity with the «Carisolv» system takes approximately 5— 15 minutes and 0.2— 1.0 ml of gel. In some cases the carious cavity's opening requires the use of a drill. Usage of a «Carisolv» system in deciduous teeth is more effective than in permanent ones due to less mineralized dentine in deciduous teeth. There are following indications for chemical and mechanical preparation of carious cavities in deciduous teeth: — class 1 (according to Ci.V. Black) carious cavities with an easy access; — class V (according to G.V. Black) carious cavities; — secondary caries; — deep caries; — fear of dental treatment, especially in children who were treated with a drill before; — inadequate behavior of a child which docs not allow to use a traditional method of treatment; — treatment of caries in children with psychoneurological disorders. Bellow there are doubtless advantages of the method: — minimal invasiveness; — maximal preservation of healthy dental tissues; — mostly absence of pain during preparation. Prepared dentine surface is uneven and provides good adhesive junction with glass-ionomer cements. Filling of carious cavities in deciduous teeth. For these purposes modern filling materials are used — glass-ionomer cements, compomers, composite materials, amalgam, in some cases — silicophosphate and zinc phosphate cements. The choice of filling materials depends on the child's age, his/her behavior at the dentist’s chair, stage of tooth development, depth and localization of carious defects. Glass-ionomer cements are widely used for deciduous teeth filling due to their adhesion to hard dental tissues as a result of chemical junction and ability to release of fluoride for some time. For filling of class I and class II carious cavities in deciduous molars it is advisable to use glass-ionomer cements of the second type (restorative), the second subtype (for loaded restorations): Ketac Molar (3M ESPE), Ketac Molar Easymix (3M ESPE). Fuji IX GP (GC) Chem Flex (Dent Splay), Jonofil Molar AC (VOCO), Kavitan (Spofa Dental), Cemion ARH (VladiMiVa). Compomers are also used for filling of deciduous teeth . They have high aesthetic properties, enough hardness. Compomers arc used with adhesive systems and don't need etching of hard tooth tissues. They include Dyract AP (DentSplay), Elan (Kerr), Compoglass (Vivadent), etc. Taking into account adhesive and anti-caries properties of

glass- ionomer cements, it is advisable to use them for filling of carious cavities of all classes in deciduous teeth during the stage of root formation of deciduous teeth. Silver amalgam with an isolating liner of zinc phosphate cement can be used for filling of class I and II cavities in deciduous teeth in older children (in root stabilization period), when there are suitable conditions for the carious cavity formation. At this stage of deciduous teeth development it is also possible to use glass-ionomer cements, composite materials and compomers. The above-mentioned materials are used for filling of class III, IV and V carious cavities in deciduous teeth. Besides, zinc phosphate cements Adhcsor (Spofa Dental, Czech Republic) and polycarboxylate cements (Poly F Plus; Adgesor Carbofine) can be used for filling of carious cavities in deciduous teeth at the root resorption stage. Treatment of deep caries in deciduous teeth Deep caries is treated in one or two visits. It is necessary to strictly follow all principles of carious cavity preparation during treatment of deep caries in deciduous teeth, as well as for permanent teeth. Softened dentine should be carefully removed from the carious cavity's walls. Only in the projection of pulp horns it is acceptable to leave a thin layer of softened dentine. After the carious cavity has been prepared it's necessary to care-fully and attentively explore it in order to exclude the case of incidentally pulp exposure. In that case treatment is provided according to the scheme of chronic fibroses pulpitis treatment. If the carious cavity's bottom is relatively hard, the pulp was not disclosed during preparation, the doctor may continue the tooth treatment. Antiseptic treatment of the carious cavity is the next stage of treatment of acute deep caries in deciduous teeth. Non-irritative broad-spectrum anti-bacterial medications (Furacilinum, Rivanolum, Ectericidum, Ethonium, Miramycinum and Microcidum) are used for that purpose. Antiseptic solution is heated to the body temperature (36-37 "C) to prevent additional pulp irritation. After the antiseptic treatment of the carious cavity is completed the doctor dries it with a warm air stream or cotton balls, and applies a therapeutic cavity liner. Pastes containing calcium hydroxide, zinc-oxide eugenol paste, and its cements are the most widely used materials for acute deep caries treatment. The doctor should keep in mind that it is not advisable to us medications stimulating dentinogenesis in the root resorption period of deciduous teeth development. Usage of self-hardening materials, like Life (Kerr), Dykal (Dent Splay), Calcimol (VOCO), allows to perform treatment of acute deep caries in one visit. In those cases, materials with high adhesive properties (glass-ionomeric cements, compomers and composites) are used for carious cavities filling. This provides the necessary filling hermeticity and prevents premature dissolution of the therapeutic cavity liner, and therefore, development of carious complications. Treatment of acute deep caries with the use of zinc oxide eugenol paste and its cements (Cariosan (Spofa Dental), IRV (Dentsply)) should be done in two visits. This is conditioned by the fact, that most permanent filling materials are incompatible with eugenol. Thus, at the first visit the prepared carious cavity is completely filled with a tough mixed zinc oxide eugenol paste or its cement. At the second visit in 1.5—3 months the doctor makes a final preparation of a carious cavity in deciduous tooth and makes a permanent filling. Chronic deep caries treatment in deciduous teeth is performed in one visit by the way of carious cavity preparation and filling. There is no need for using of special cavity liners for the cavity bottom. Filling materials are used according to the stage of the deciduous tooth development (stabilization, resorption). Hydroxyapatite is formed by 8—9 atoms of Ca2+ instead of 10 atoms. One or two of these atoms are replaced by hydrogen (H) or hydroxonium (I bO') ions. Thus, hydroxyapatite is not destroyed, but its ability to resist to acids lowers with decreased calcium content and decreased calcium- phosphorus ratio of enamel. Further formation of organic acids on the enamel surface leads to further demineralization and gradual extension of microcavities between the crystals of enamel prisms. As a result, the abovementioned process creates favorable conditions for penetration of microorganisms and their metabolic products into enamel microdefects. Thus, the source of acid formation moves into the enamel. At this stage of the carious development, the enamel demineralization spreads both along the surface and in depth, making a cone-shaped focal lesion.

More resistant superficial enamel layer gets dissolved in long-term uncured demineralized segments; that leads to defect formation. As a result, initial caries becomes superficial clinically. Development of dental caries is possible under the influence of cariogenic microflora in a certain conditions only. The following factors are pathogenetic for dental caries: dental resistance, content and properties of saliva, eating habits (V.K. Leontiev, 1994).

## TOPIC 47.

## DIFFERENTIAL DIAGNOSIS OF ACUTE PULPITIS.

Pulpal hyperemia is an initial stage of acute inflammation of pulp. It should be noted that an acute inflammation of pulp typically develops in a closed pulp camber, which determines the clinical picture of the disease.

Complaints, hyperemia of pulp is characterized by short spontaneous acute pain which can be provoked by a cold thermal irritants. Pain attack lasts for 1—2 minutes and is replaced by a painless interval for 12—48 hours. The pain is localized. Objective examination defines a deep carious cavity, but in teeth with immature roots the cavity is relatively less deep. Walls and bottom of the cavity contain softened light or slightly pigmented dentine. Probing of a carious cavity is slightly painful. Cold thermal irritants provoke a severe pain lasting for 1—2 minutes. Pulpal hyperemia is more often detected in permanent teeth with formed roots in somatically healthy children.

Pathological anatomy. Microscopically pulpal hyperemia is presented by an dilated capillary network, injected vessels, mainly in the area of pulp horns. The vascular picture is clearly expressed, the leukocytes are marginalized; the pulp is slightly swollen.

Differential diagnosis. Pulpal hyperemia should be differentiated from acute deep caries and acute serous localized pulpitis. Presence of spontaneous pain and pain that is provoked by cold thermal irritants and lasts for 1—2 minutes after the irritant's removal differentiates pulpal hyperemia from acute deep caries. In case of acutc serous localized pulpitis, unlike pulpal hyperemia, spontaneous paroxysmal pain lasts more (15-30 minutes) and occurs mainly at night.

Acute serous localized pulpitis. In case of acute serous localized pulpitis inflammation covers the coronal pulp or its part adjacent to the carious cavity. Children complain of acute pain which occurs mainly at night. Initially pain attacks last for 15—30 minutes with long remissions, but with the development of the inflammation their duration increases to 40—45 minutes. The intervals between the first pain attacks last 2—3 hours, but later they get shorter. Pain is localized; child can correctly identify the painful tooth. Low temperature irritants provoke a pain attack lasting from 30 minutes to 1-2 hours. Cold food, drinking and mouth rinsing (temperature of water 22—26 "C) cause pain attacks. Objective examination defines a carious cavity corresponding acute deep caries. The cavity bottom is covered with light softened dentine which is removed by layers. The carious cavity is not connected with the pulp chamber; sometimes, a pulpal horn can be seen through its bottom. Probing is painful, especially in the limited area of projection of the pulpal horn. Electrical excitability of pulp is about 20pA in the tooth with developed root. Acute localized pulpitis usually does not last more than 2 days. Pathological anatomy. Dilated vessels in the coronal pulp are overloaded with blood; punctual hemorrhages are seen. The pulp chamber is filled with serous exudates; there are clusters of neutrophilic leukocytes. I n a limited area (around the pulp horn) there is a cluster of neutrophils, lymphocytes and monocytes. There are degenerative changes of odontoblasts in the pulpal peripheral layer adjacent to the carious cavity.

Differential diagnosis. Acute serous localized pulpitis should be differentiated from acute diffuse pulpitis and aggravation of chronic fibrous pulpitis. In case of acute diffuse pulpitis pain attacks last up to several hours with short remissions (less than 30 minutes); pain is irradiating; percussion becomes painful. In case of aggravation of chronic fibrous pulpitis acute pain attacks may have occurred before. Objective examination defines a connection between the carious cavity and the pulp chamber practically in all cases

Acute serous diffuse pulpitis is a result of further development and extension of acute inflammation to the root pulp. In this case the clinical picture significantly changes. Children complain of spontaneous acute pain irradiating along the trigeminal nerve. Pain lasts for hours. That proves the development of diffuse pulpitis, which is characterized by the inflammation of the coronal and root pulp. Pain continues for several hours with short remissions; child practically can't sleep at night. The low temperature irritants provoke an intensive pain attack. Pain irradiation is one

of the features of acute serous diffuse pulpitis. In case of pulpitis in maxillary teeth pain irradiates along the second branch of the trigeminal nerve; in case of pulpitis in the mandibular teeth - along the third branch. In case of pulpitis in frontal teeth pain irradiated to the opposite side of the jaw. In teeth with immature roots the pain is less intensive, it does not irradiate, and pain attacks are shorter and less intensive. Diffuse form of pulpal inflammation in the tooth with immature roots can develop within a day. Objective examination defines a deep carious cavity separated from the pulp chamber with a thin layer of softened dentine. Low temperature irritants provoke a acute long-lasting pain. Probing is accompanied by significant pain sensations over the bottom of a carious cavity. Pain from vertical percussion is a specific objective symptom. This symptom is a result of development of perifocal periodontitis and it is a feature of a diffuse pulp inflammation. Electrical excitability of pulp is about 40—50 pA. Pathological anatomy. Blood vessels are dilated and injected. The leukocytes are marginalized, neutrophilic leukocytes migrate through the walls of the vessels that results into local infiltration and accumulation of polymorphonuclear leukocytes. Damages of pulpal vessel, minor hemorrhages and edema of pulpal tissue are observed. Features of degenerative changes are identified in the layer of odontoblasts in the coronal pulp. Differential diagnosis. Acute serous diffuse pulpitis should be differentiated from acute serous localized pulpitis and acute purulent pulpitis. In case of acute serous localized pulpitis the pain is localized, the pain atacks are short, they intensify from low temperature irritants; the periods of remission are long. In case of acute purulent pulpitis pain is permanent; it intensifies from hightemperature influences and decreases from low-temperature influences; percussion is very painful; regional lymph nodes are often enlarged. Sometimes the child’s body temperature increases.

Acute purulent pulpitis develops as a result of localized or diffuse serous inflammation. Child

complains of spontaneous pulsating almost permanent pain. Its intensity increases, and some time it subsides, and the child cannot clearly indicate the painful tooth. At night pain becomes more intensive, exhausting. It increases from high-temperature irritants (more than 37 °C). Low temperature somewhat weakens the pain attack. In a tooth with immature roots pain is less intensive and it does not irradiate along the trigeminal nerve. Objective examination defines a deep carious cavity with softened dentine on the bottom. The superficial probing is painless. Opening of the pulp chamber reveals a drop of pus or blood-purulent exudate. Deep probing is painful. After opening of the pulp chamber pain intensity decreases abruptly. In case of spontaneous opening of the pulp chamber inflammation may become chronic. Percussion of the tooth is very painful, which indicates the presence of perifocal periodontitis. Acute purulent pulpitis in children is often accompanied by the transition of inflammation to soft tis-sues, which is proved by a collateral edema, increased and painful regional lymphatic nodes. These changes are more common in case of acute purulent pulpitis in teeth with immature roots. Pathological anatomy. Pulpal blood circulation is severely damaged; significant migration of neutrophil leukocytes and erythrocytes from blood vessels arc observed. Purulent dissolution of tissues of various sizes (pulpal abscesses) is observed. Differential diagnostics. Acute purulent pulpitis should be differentiated from acute serous pulpitis and acute purulent periodontitis. In case of acute serous diffuse pulpitis pain is paroxysmal and irradiating; it mostly occurs at night, increases from low temperatures and decreases from high. In case of acute purulent periodontitis pain is permanent, pulsating and increasing; tooth does not react to thermal irritants, the pulp is destructed in the root canals, probing is painless. Tooth percussion is painful; soft tissues around the tooth are significantly inflamed.

Acute traumatic pulpitis in children is a common case. Development of acute traumatic pulpitis is associated with a pulp injury occurring during preparation and formation of a carious cavity, or as a result of fracture of tooth crown. Mechanical pulp injury is usually accompanied by

its infcetioning. Pulp horn exposure during preparation of the carious cavity more often occures in case of acute deep caries. Acute pain and a drop of blood are the first features of the pulp injury. Pain increases from probing. If the perforation hole is well visible, the doc-tor should not probe the opened pulp to avoid its further injury and infectioning. There is a small hole in the carious cavity bottom, and a bright red pulp is visible. Pulp exposure in case of tooth crown fracture results from an acute injury (blow, bruise). In case af a traumatic tooth crown injury (without pulp exposure) an acute pulpitis with a post-traumatic necrosis can develop; it is diagnosed in 6—7 days after the injury. Pathological anatomy. Changes in pulp correspond to the acute serous pulp inflammation (localized or diffuse). The pulp structure is damaged. first of all the odontoblast layer is affected. Hyperemia, exudation, single hemorrhages or a more extensive hematoma, penetration of dentine fragments into the pulp are observed.

## TOPIC 48.

## DIFFERENTIAL DIAGNOSIS OF CHRONIC PULPITIS.

Chronic fibrous pulpitis is the most common form of pulpitis in permanent teeth at any stage of root development. Chronic fibrous pulpitis in children can develop as a primary chronic process without the preceding stage of acute inflammation, which is a specific feature of this form of pulpitis. Children complain on pain in case of mechanical irritation (food elements, bristles of tooth brush). Pain is immediately relieved after the irriant's removal. Another typical feature is pain caused by temperature changes. However, chronic fibrous pulpitis in children may progresses asymptomatically, and may only be detected at the regular oral cavity sanation. A spontaneous pain is not typical for chronic fibrous pulpitis; it is a symptom of aggravation of a chronic inflammatory process. Objective examination defines a deep carious cavity connected with the pulp chamber; the pulp is dark-red, bleeding and painful from probing. Chronic fibrous pulpitis in children may also develop with a closed pulp chamber, especially at the stage of root formation. In this case the bottom of the carious cavity is covered with softened slightly pigmented dentine. In case of prolonged course of chronic fibrous pulpitis the radiological survey detects destructive changes in the periapical tissues. In this case, chronic fibrous pulpitis is complicated with periodontitis. Pathological anatomy. Productive processes prevail and it leads to partial or complete pulp fibrosis. The pulp contains a small quantity of specialized cell elements. In root pulp odontoblasts can sometimes be pre-served. In teeth with immature roots metaplasia of pulp into a solid connective tissue is less expressed.

Differential diagnostics. Chronic fibrous pulpitis should be differentiated from acute and chronic deep caries, chronic gangrenous pulpitis and chronic periodontitis. In case of deep caries pain is never caused by food stuck in the carious cavity, caries progresses asymptomatically, and there is no connection of the carious cavity with the pulp chamber. In case of chronic gangrenous pulpitis, unlike chronic fibrous pulpitis, pain is provoked by high temperature irritants, pulp probing is painful in root canals only; destructive changes in periodontal tissues are very frequent. In case of chronic periodontitis pulp chamber exposure and entering the root canals are painless

Chronic hypertrophic pulpitis is one of the forms of productive pulp inflammation; it is characterized by growth of granulating and young connective tissues in it. Children complain of bleeding «from the tooth» and, very rarely, of pain during eating and tooth brushing. Sometimes the doctor can find out from the anamnesis that the tooth has hurt before. Objective examination defines a carious cavity filled with epithelialized granulating tissue, which has a shape of a red smooth polyp on a wide neck; granulating tissue fills almost the entire carious cavity. Its surface is slightly sensitive, probing causes pain and slight bleeding. Pathological anatomy. Hyperplastic processes prevail in the coronal pulp; there is considerable expansion of young connective and granulating tissues with large number of fibroblasts, histiocytes and lymphocytes. Odontoblasts in the coronal and root pulp are degeneratively changed. Differential diagnostics. Chronic hypertrophic pulpitis should be differentiated from hyperplasia of interdental papilla and its ingrowth into the carious cavity and from granulating tissue that ingrown through a perforation in the bottom or the wall of pulp chamber, which is usually a result of the destruction of tooth bifurcation. A radiological survey will help to solve a problem of differential diagnosis. The final diagnosis should be made basing on the analysis of radiogram.

Chronic gangrenous pulpitis. This form of pulpitis is characterized by complete or partial necrotization of the coronal pulp and localization of the chronic inflammation process in the root pulp.

Complaints.Sometimes patients complain on pain from hight-tempcrature irritants or unpleasant smell from oral cavity (foeter ex ore). Sometimes the doctor finds out from the anamnesis toothache before. Objective examination defines a carious cavity which is often connected with the pulp chamber. Probing of the connection with the pulp chamber is painless. Pain and bleeding start after probing of the orifices of root canals. Sometimes, painful bleeding pulp is detected at one root canal orifice, and painless necrotized pulp — in other canals of a multiroot tooth. In case of this form of pulpitis the radiogram shows destructive changes in periapical tissues indicating the presence of pulpitis complicated with focal periodontitis. Pathological anatomy. Pulp structure is completely destroyed in its coronal part: unstructured mass, granular dissolving, colonies of microorganisms. The root pulp part contains areas of necrosis, fibrosis and growing granulating tissues. Differential diagnosis. Chronic gangrenous pulpitis should be differentiated from chronic granulating periodontitis and chronic fibrous pulpitis. In case of chronic granulating periodontitis the radiogram defines destructive changes in the periapical tissues. Clinically a fistula is often defined on mucosa of an alveolar process.

Chronic concremental pulpitis. The causative factor of this form of pulpitis are denticles — calcified deposits in the dental pulp. Usually they are located in the pulp chamber or root canals. It may be composed either of irregular dentine (true denticle) or ectopic calcification of pulp tissue. Denticle is formed in teeth, located in the back (molars, premolars), most often in persons over 40 years. These formations cause the permanent irritation of nervous endings of pulp, resulting in chronic inflammation.

Patients complain of spontaneous pain and development of acute pain attack, referred from one arch to the other and along the branches of n. trigeminus. The character of pain attack is like of neuralgic attacks. Pain attacks increase and become more frequent at night and under vibration. Clinical features resemble trigeminal neuralgia. Pain attack lasts 15-30 minutes. Vertical percussion is painful and may provoke pain attack.

Exacerbation of chronic pulpitis. Every form of chronic pulpitis may be exacerbated. Often this is fibrous pulpitis, more rarely — chronic gangrenous pulpitis. Exacerbative chronic pulpitis is characterized by spontaneous pain and development of acute pain attacks, referred from one arch to the other and along the branches of n. trigeminus. Pain attacks increase and become more frequent at night and at horizontal position of patient. The pain attack can be provoked by different irritants, usually the cold. Duration of pain attack may be different: at first it lasts 15-30 minutes, but with development of the inflammatory process in the pulp its duration increases to 1-2 hours. When inflammation develops in root pulp, vertical percussion becomes painful. Pulpitis complicated apical periodontitis. The clinical features combined clinical signs of pulpitis and apical periodontitis. For example it may be signs of acute diffuse or purulent pulpitis and signs of apical periodontitis. The durability of pain attack was 10-30 minutes and now attacks last about an hour. The durability of painless intervals decreases to 10-30 minutes. Pain attacks increase and become more frequent at night and at horizontal position of patient. Usually patients indicate on the causal caries tooth.

These signs of pulpitis combine with signs of apical periodontitis. There can be edema of soft facial tissues (lips, cheeks), especially on the site of affected tooth. There is hyperemia and edema in the area of tooth apex, sometimes hyperemia is present in the gums surrounding areas. In some cases the periosteal abscess may develop. Horizontal and vertical percussion of the tooth is very sensitive. The tooth mobility in medial, distal and vertical directions is observed.

**Topic 54. Dystrophic changes in the pulp in general diseases of the body.**

Degenerative-(dystrophical)-inflammatory and degenerative diseases 1. Generalizative parodontitis Tendency: chronical, exacerbation, remission. Degree: initial, I, II, III. Expension:

diffuse lesion of parodontium. 2. Parodontosis Tendency: chronical. Degree: initial, I, II, III. Expension: diffuse lesion.

Pulpitis. In all forms of pulpitis, the patient's face is symmetrical, regional lymph nodes are not enlarged, but may be painful when pulpitis is complicated by periodontitis. Traumatic pulpitis: a) the pulp is accidentally opened in the treatment of caries. During the preparation, the patient experienced a sharp, instant pain. On examination of the carious cavity there is a red pulp at one point, extremely tender on touch and chemical or temperature stimuli. In the case of incomplete exposure, when the pulp is covered by a thin layer of predentine or demineralized dentine, the exposure point is not visually determined. The unnoticed-by-the-curator exposed pulp, when 4 applying the insulating liner, will cause increasing pain to the end of filling of the carious cavity; b) exposure of the pulp as the result of the crown fracture of the front teeth and the cuspal fracture (buccal and lingual) of premolars and molars. On the plane of fracture the exposed pulp, which is extremely tender on temperature and chemical stimuli, can be detected with a probe or visually, EPT (Electric Pulp Testing) is 6-12 μA. Hyperemia of the pulp. In the area of the affected tooth, the transitional fold is unchanged in color, painless. In the tooth there is a filling or a deep carious cavity (specify class) with signs of acute or chronic caries, probing the bottom of the carious cavity is painful in the projection of the pulp horn. The reaction of the tooth to chemical and temperature stimuli causes an attack of localized pain lasting for several minutes. The tooth percussion is painless, the electrosensitivity is reduced (15-20 μA). In acute partial pulpitis, the above signs increase. Acute general (diffuse) pulpitis. In the tooth there is a filling or a carious cavity with signs characteristic of acute or chronic caries. Probing is painful throughout the cavity floor, indicating demineralized dentin. Reaction to chemical irritants and to temperatures of 30° C and below, as well as of 40° C and higher causes an attack of acute pain irradiating into the corresponding area. The attack can last for several dozens of minutes and is removed by painkillers. Percussion is painless or unpleasant; the electrosensitivity of the pulp is reduced (35-40 μA). In the area of the affected tooth the transitional fold is unchanged in color, painless. Acute purulent pulpitis. The face is symmetrical, regional lymph nodes are not palpable. In the tooth there is a filling or a deep carious cavity, the probing of the bottom is painless, in the process a droplet of purulent secretion can be produced with synchronous reduction of the pain severity. The temperature of 36° and above causes an attack of pulsating pain irradiating into the corresponding area; the temperature below 35° C relieves the pain. The reaction to percussion is in the form of mild pain or discomfort. EPT is 40-50 μA. The transitional fold near the affected tooth is either unchanged or hyperemic in color, painless on palpation. Chronic simple (fibrous) pulpitis. The face is symmetrical, the regional lymph nodes are not enlarged. In the tooth there is a filling or a deep carious cavity with signs of acute or chronic caries. The tooth cavity can be: a) open; exposed pulp is pink, tender on touch; b) not open; probing is painful all over the bottom of the carious cavity. Chemical irritants and temperatures of 30° and below, as well as of 45° and above cause a reaction of dull pain, rarely of acute pain, which slowly ceases after the irritation is eliminated. Percussion is painless or slightly painful. EPT is 35-50 μA. The transitional fold near the affected tooth is unchanged in color, painless. Chronic hypertrophic pulpitis. The face is symmetrical, the regional lymph nodes are not enlarged. In the tooth there is a carious cavity, partially or completely filled with pink or gray pulp tissue with a smooth or lobular surface. Exuberant granulation tissue is slightly tender and bleeding on probing. The cold and chemical stimuli cause non-acute pain, which lingers after elimination of the 5 stimulus. The tooth's response to percussion is painless or slightly painful. The transitional fold near the affected tooth is unchanged in color, painless. Chronic gangrenous pulpitis. The tooth is grey, the carious cavity in most cases connects with the tooth cavity, but also may not be open, contains a stinking dark homogeneous mass. Pain is caused only by deep probing. The reaction to percussion can be painful, the temperatures of 40-50°C cause pain and heaviness, which slowly cease after the stimulus is removed. EPT is 60-80 μA. The X-ray image for all forms of pulpitis is

more often characterized by the absence of radiographic changes, however, sometimes changes that are characteristic of chronic fibrous periodontitis can be observed. Chronic concremental pulpitis. The face is symmetrical, regional lymph nodes are not palpable. On examination of the tooth, you can find indirect signs of concremental pulpitis, namely: pathological abrasion, wedge-shaped defect, a carious cavity, a filling. The anamnesis can reveal that the tooth was treated for caries or pulpitis using odontotropic pastes. Tooth percussion can cause a feeling of heaviness, rarely an attack of pain when the concrement is not attached to the wall of the tooth cavity. Chemical and temperature stimuli do not cause a reaction from the pulp. The electrosensitivity of the pulp is reduced. The transitional fold is unchanged in color, responds negatively to palpation. The certain symptom of the disease is the presence of concrements (denticles or calcifications) in the tooth cavity on an X-ray. Denticles have clear contours, often with sharp projections. The X-ray density of the denticle is the same as that of the dentin in the wall of the tooth cavity. The tooth cavity, in the part where it is not filled with the denticle, has clear contours, it is traced around the denticle as a continuous or fragmented area with a higher radiability (on an X-ray it is of a darker color). Calcifications are determined in the cavity of the tooth as dense formations, but their radiability is slightly lower than that of dentin. There are no clear contours and sharp projections. The tooth cavity is somehow veiled, its contours in the area of the calcification are blurred.

## TOPIC 49.

## DIFFERENTIAL DIAGNOSIS OF PERIODONTITIS.

CLINICS OF PERIODONTITIS IN PERMANENT TEETH Chronic infectious periodontitis in permanent teeth is the most frequent periodontium disease in children. Chronic inflammation in periodontium can start as a result of acute inflammation; however, in teeth with immature roots it is more frequent as a primarily chronical process. Granulating form is the most common form of chronic periodontitis in permanent teeth in children, especially at the root formation stage. Chronic granulating periodontitis is the most widespread form of chronic periodontitis in children. Pathomorphological picture. The histological research in the center of chronic inflammation predominantly defines young understructured connective tissue, neogenic capillaries and nerve fibers, as well as a significant amount of fibroblasts, macrophages and plasmocytes. Along with growth of granulations, an intensive osteoclast lacunar resorption of bone tissues, cements and dentine in the causative tooth roots is observed. Clinics. As a rule, chronic granulating periodontitis develops without pain symptoms. Children visit a dentist with complaints on a tooth color change, or presence of a fistula with purulent excretion. The doctor defines a filling or a carious cavity in the causative tooth during the objective examination. Probing of the carious cavity bottom is painless. The probing can often detect a painless connection with the pulp chamber. In case of chronic granulating periodontitis in permanent teeth with underdeveloped teeth an ingrowth of granulating tissues into the root canals from the periapical destruction segment is often observed. In this case deep probing is slightly painful and is accompanied by bleeding. Fistula is the main clinical feature of this form of chronic periodontitis in permanent teeth in children. Gingival mucosa is slightly swell and congestively hyperemic; it has cyanochroic coloring. The granulating form of chronic periodontitis in permanent teeth in children may be accompanied with regional lymphadenitis. Development of chronic granulating periodontitis in immature permanent teeth is complicated with destruction of the growth zones and termination of further root formation. Radiologically chronic granulating periodontitis is characterized by destruction of an alveolar cortical plate and presence of a resorption (enlightening) area with indistinct contours in a spongiose bone tissue in the periapical root area. Bone tissue destruction can also be observed in the bifurcation area of permanent molars owing to: penetration of infection and the pulp destruction products via additional canaliculi of the pulp chamber bottom (especially in immature teeth); or the pathological process diffusion from the periapical area. The radiological picture of chronic granulating periodontitis in immature permanent teeth should be differentiated with and intact growth zone. Integrity of the cortical plate around the growth zone (enlightenment segment of the bone tissue), indicates on the absence of pathological process in this area. Chronic granulating periodontitis in permanent teeth in children should be differentiated with chronic deep caries, chronic fibrous and gangrenous pulpitis, and pulpitis complicated by a focal periodontitis. The final diagnosis of chronic granulating periodontitis should be based on the following data: clinical examination (fistula with granulations and purulent excretion; fistula scar; swell and hyperemic gingival mucosa; tooth color change), and the radiological results (alveolar cortical plate destruction; bone tissue resorption area with indistinct contours). Chronic granulomatous periodontitis in permanent teeth in children occurs predominantly in the period of completely developed roots. Pathomorphological picture. The microscope examination defines the growth of granulation tissues. The growth is limited from health bone tissues with a connective tissue capsule. Its fibers are twisted into periodontium, thus the granuloma is tightly connectcd with the tooth root. The granuloma center contains fibroblasts, lymphocytes, plasmocytes and tissue basophils located randomly. Most granulomas contain single epithelial cells or their cords. The bone tissue around the capsule is dense, thus the lesion center has distinct contours on the radiogram. Clinics. Chronic granulomatous periodontitis in permanent teeth in children is predominantly has symptomless

clinics. However, some patients may complain of unpleasant sensations at applying pressure on the causative tooth, and its color change. The tooth may be intact (in case of traumatic periodontitis), filled or it may have a carious cavity communicated with the pulp chamber. Probing of a cavity bottom, its communication with the pulp chamber and the root canal orifices is painless. The tooth percussion is painless; there is no reaction to thermal irritants. The diagnosis of chronic granulomatous periodontitis is based on radiological examination results. Destruction the alveolar cortical plate and a dissolved bone tissue area of a round or oval shape with distinct contours (5 mm in diameter) is observed in the root apex area. Chronic granulomatous periodontitis in children should be differentiated from the growth zone of intact immature teeth. Radiological features of the growth zone: integrity of the alveolar cortical plate around the growth zone; regular width of the periodontal fissure near the developed root part. Chronic granulomatous periodontitis should be differentiated from the following diseases: — chronic deep caries, characterized by a pain symptom during preparation of enamel-dentine junction, and the tooth reaction to low- temperature irritations; — chronic fibrous and gangrenous pulpitis complicated by focal periodontitis; they are characterized by a sharp pain at probing of the carious cavity communication with the dental cavity and the root canal orrifices; — chronic granulating and fibrous periodontitis (by a radiological examination). In case of granulating periodontitis the bone tissue resorption area does not have distinct contours. The fibrous form is characterized by deformation of the periodontal fissure and the integrity preservation of the alveolar cortical plate. — cystic granuloma and a radicular cyst: On the radiogram the bone destruction area has a diameter of 5—8 mm. Chronic fibrous periodontitis in permanent teeth in children is rarely diagnosed as compared to other forms of chronic periodontitis. It is characterized by formation of a coarse-fibered connective tissue in the apical root part. This tissue replaces periodontium. Some authors interpret these periodontium changes as fibrosis and do not consider the process as inflammation. Fibrous periodontitis can develop in permanent teeth with formed roots as a result of an acute periodontium inflammation, more often - of traumantic origin. Sometimes fibrous periodontitis is observed in teeth treated for pulpitis before, or as a favorable outcome of an effective treatment of other chronic periodontitis forms (granulating, granulomatous). Pathomorphological picture. The microscopic picture is characterized by a diffusive swelling of periodontium and its transformation into a coarse-fibered connecting tissue. Lymphocytic infiltrates are defined in some segments. Sometimes, the periodontal fissure can be narrowed due to hypercementosis — the secondary cement formation all over the root surface or only on one side of the root. Clinics, fibrous periodontitis is characterized by a symptomless course, complaints of pain are absent. Objectively: the tooth is intact (in case of traumatic origin), or filled; more rarely — a carious cavity is detected. Percussion is painless. The radiogram shows a deformation of the periodontal fissure as an uneven expansion or narrowing in the hypercementosis zones. The radiological semiology of fibrous periodontitis is similar to features of teeth with immature roots. At the stage of open apical foramen and immature periodontium, the periodontal fissure is dilated, especially in the root apical part. For the final diagnosis it is necessary to consider the child's age and the duration of root growth in various groups of teeth. Acute periodontitis in permanent teeth in children usually develops as a result of acute dental trauma (blow, falling) or is a consequence of errors in endodontic treatment of pulpitis. Development of acute toxic periodontitis, especially in teeth with immature roots, is caused by the use of pastes containing arsenic for pulp devitalization. It can also be caused by the use of the fenol group of medications (phenol, camphorated phenol, tricresol, pheresol, resorcin) and aldehydes (formalin) for antiseptic processing and filling of root canals. Acute periodontitis of infectious genesis in permanent teeth in children often begins as a perifocal process in case of acute diffuse and purulent pulpitis. Pathomorphological picture of acute serous periodontitis: microscopic examination reveals periodontium vessels' expansion and their increased permeability; edema and infiltration of its tissues with neutrophilic leukocytes and (in small number) with macrophages and lymphocytes.

Clinics of acute serous periodontitis. Patients complain of constant increasing pain in the causative tooth and a feeling of «an evolved tooth». The pain increases at biting, therefore children practically do not use the affected side during meal. The patients' general condition docs not change much. In case of traumatic origin of periodontitis the tooth is intact, or it may have a break-of in the crown part at varying levels (enamel / enamel and dentine). In case of acute toxic periodontitis there are features of partial preparation of carious cavity, partial or complete pulp chamber disclosure. In case of acute periodontitis of infectious genesis there is a caries cavity, which is not connected (as a rule) with the pulp chamber. In case of pulp destruction (necrosis) and the periodontium focal process development, the carious cavity probing is painless. There is no reaction to thermal irritation. Vertical percussion couses acute pain. The tooth may be slightly mobile due to exudate accumulation in periodontium. Gingival mucosa around the causative tooth is unchanged, or it may have insignificant inflammatory features; it may be pastose, slightly hyperemic and slightly painful at palpation. Regional lymphatic nodes are sometimes enlarged, slightly painful, but more often they are not palpated. There are no radiological changes in periodontium in case of acute serous inflammation. It should be noted, that in case of acute periodontitis in permanent teeth in children the process gets a diffuse character rapidly, the serous inflammation phase may pass into the purulent one within a day. Pathomorphological picture of acute purulent periodontitis: microscopic examination reveals edema; increase of periodontal tissue infiltration with polymorphonuclear leukocytes; accumulation of purulent exudate; and dissolution of fibrous tissues. Clinics of acute purulent periodontitis is characterized by a constant intensive throbbing pain. Even a slight touch with tongue or a tooth- antagonist provokes an acute pain; therefore patients keep their mouths half-opened. Hypersalivation is possible. Pus expansion under periostenum may relief pain. The patients' general condition worsens owing to effervescence and intoxication development. Other symptoms include general asthenia, headache, and sleep and appetite disturbance. Objectively, the tooth may be intact, treated before or it may have a caries cavity which is not connected with the pulp chamber. An intensive constant pain, increasing at vertical and horizontal percussion, is the main clinical feature. Diffuse expansion of the process causes pain at the adjacent teeth's percussion. The gingival mucosa in the inflammation segment is brightly hvperemic, swell and painful at palpation. As a result of purulent exudate expansion under periostenum, an abscess is formed; it is characterized by a flattened mucosa fold in the causative tooth area, painful palpation, and, sometimes, a fluctuation symptom. In a number of cases the acute purulent periodontitis causes facial asymmetry clue to collateral edema of soft tissues. Submandibular lymph nodes are enlarged, dense, and painful at palpation. Radiological changes are absent in most cases of acute periodontitis. However, in some cases the sharpness of the spongiform bone substance contours may be lost due to diffuse pus expansion. Acute purulent periodontitis should be differentiated with the following diseases: — acute diffuse pulpitis complicated by perifocal periodontitis, which is characterized by spontaneous paroxysmal pain. Probing of a carious cavity is painful all around the cavity bottom. Pulp chamber exposure is accompanied by an intensive pain and bleeding. Patients'general condition is practically unchanged; — chronic periodontitis exacerbation — basing on the radiological examination results (destructive changes in the periapical tissues); — acute odontogenous periostitis. Objectively: the mucosa fold near the causative tooth and the adjacent teeth is flattened, swell, hyperemic and painful at palpation. — acute odontogenous osteomyelitis. Objectively: mobility of the causative tooth and adjacent teeth; flattened mucosa folds from both vestibular and oral sides of the alveolar process; pus excretion from dentogingival pockets. Aggravation of chronic periodontitis in permanent teeth with immature roots in children is much more often diagnosed than its acute course. Clinics of the chronic inflammation process' aggravation is similar to that of acute periodontitis. The following clinical features are used for differential diagnosis of the aggravation: changed color of the tooth; presence of a functioning fistula or its scar; carious cavity connection with the pulp chamber, mainly in teeth with mature

roots. The history may include previous aggravations of the pathological process. The aggravated course is characterized by the following radiological features: destruction of the alveolar cortical plate; presence of the bone resorbtion area with indistinct contours and deformation of the adjacent periodontal fissure. Differencial diagnostics between the aggravated and acute periodontitis should consider the previous (if any) aggravations in the history; presence of a fistula or its scar; the tooth's color change, and destructive changes in periodontium. Acute regional (marginal) periodontitis develops as a result of mechanical damage of a gingival margin and a circular ligament (at toothpick use, tool damages during the dental treatment) with further penetration of infection. It may also result from the use of chemical substances (acids, alkalis) or devitalizing pastes. Marginal periodontitis may also he caused by a foreign substance penetration, traumatizing by a matrix or an over-hanging edge of a filling. Clinics of an acute marginal periodontitis: the child complains of constant pain in the affected tooth area. The gingival edge is hyperemic swelled and painful at horizontal percussion. In case of purulent inflammation a painful infiltrate is formed, pus excretes from the dentogingival pocket. The purulent exudate accumulation may lead to formation of a subgingival abscess. Radiological examination does not define any bone tissue destructive changes; however, it may detect a filling with overhanging edges or a foreign substance. As a rule, chronic marginal periodontitis develops as a result of long lasting influence of a mechanical (injuring) factor. The patient may complain of insignificant painful sensations of aching character. An objective examination determines moderate puffiness, congestive hyperemia and cyanosis of a marginal gingiva. Retraction of gingival papillaes is observed sometimes. Horizontal percussion is slightly painful. Ragiological examination defines an overhanging filling edge, expansion of a periodontal fissure in the causative area; in case of a long lasting course — resorption of a cortical plate and spongious bone at tops of interdental septs. Clinics of aggravated chronic marginal periodontitis is similar to its acute form’s symptoms. The radiological examination reveals destructive changes characterizing chronic marginal periodontitis.

## TOPIC 50.

## X-RAY DIAGNOSIS OF PERIODONTITIS.

Radiologically chronic granulating periodontitis is characterized by destruction of an alveolar cortical plate and presence of a resorption (enlightening) area with indistinct contours in a spongiose bone tissue in the periapical root area. Bone tissue destruction can also be observed in the bifurcation area of permanent molars owing to: penetration of infection and the pulp destruction products via additional canaliculi of the pulp chamber bottom (especially in immature teeth); or the pathological process diffusion from the periapical area. The radiological picture of chronic granulating periodontitis in immature permanent teeth should be differentiated with and intact growth zone. Integrity of the cortical plate around the growth zone (enlightenment segment of the bone tissue), indicates on the absence of pathological process in this area. Chronic granulating periodontitis in permanent teeth in children should be differentiated with chronic deep caries, chronic fibrous and gangrenous pulpitis, and pulpitis complicated by a focal periodontitis

The diagnosis of chronic granulomatous periodontitis is based on radiological examination results. Destruction the alveolar cortical plate and a dissolved bone tissue area of a round or oval shape with distinct contours (5 mm in diameter) is observed in the root apex area.

chronic granulating and fibrous periodontitis (by a radiological examination). In case of granulating periodontitis the bone tissue resorption area does not have distinct contours. The fibrous form is characterized by deformation of the periodontal fissure and the integrity preservation of the alveolar cortical plate. — cystic granuloma and a radicular cyst: On the radiogram the bone destruction area has a diameter of 5—8 mm fibrous periodontitis is characterized by a symptomless course, complaints of pain are absent. Objectively: the tooth is intact (in case of traumatic origin), or filled; more rarely — a carious cavity is detected. Percussion is painless. The radiogram shows a deformation of the periodontal fissure as an uneven expansion or narrowing in the hypercementosis zones. The radiological semiology of fibrous periodontitis is similar to features of teeth with immature roots. At the stage of open apical foramen and immature periodontium, the periodontal fissure is dilated, especially in the root apical part. For the final diagnosis it is necessary to consider the child's age and the duration of root growth in various groups of teeth.

## TOPIC 51.

## ERRORS AND COMPLICATIONS IN THE DIAGNOSIS OF PULPITIS AND PERIODONTITIS.

Groups of mistakes:

Mistakes during diagnostic

During treatment

After treatment

Mistakes during diagnostic:

Not correct diagnosis

Wrong tooth diagnosed

Mistakes during treatment:

-not totally disinfected root canal

- overwiding of root canal

- trauma of apex

- Fracture of instrument

- Perforation of wall of root

- perforation of apex

- perforation of root bifurcation

- Overfilling of root

- not complete filling of root

Mistakes after treatment:

-postoperative pain

- apical periodontitis

- resorbtion of bone

## TOPIC 52.

## MODERN TECHNOLOGIES FOR THE TREATMENT OF PULPITIS.

1. Instruments for use during access cavity preparation (a) Basic instrument packs (b) Burs (c) Rubber dam. 2. Instruments for use during root canal preparation(a) Hand instruments (i) Barbed broaches (ii) Reamers (iii) Files: a. K-type b. K-flex c. Flexofile d. Flex-R Basic instrumentation in endodontics e. Hedstroem f. S-File (b) Power-assisted root canal instruments(i) Reciprocating handpieces (ii) Ultrasonic instrumentation (iii) Sonic instrumentation (iv) The SET canal finder system (c) Electronic canal measuring devices (d) Measuring instruments, gauges and stands (e) Instruments for the retrieval of broken instruments and posts - The Masserann kit 3. Instruments used in the filling of root canals (a) Lateral and vertical condensation (b) Thermomechanical compaction (c) Thermoplasticized injectable gutta-percha (d) Obtura system (e) Ultrafil system (d) The Endotec thermal endodontic condenser (e) Spiral root canal fillers 4. Equipment for storage and sterilization of instruments

## TOPIC 53.

## MODERN TECHNOLOGIES OF TREATMENT OF PERIODONTITIS. PHYSICAL FACTORS IN THE TREATMENT OF COMPLICATED CARIES.

Treatment of apical periodontitis

The methods of apical periodontitis treatment can be divided into 4 groups:

1.conservative — aimed at preserving anatomic and functional value of the affected tooth;

2.conservative-surgical — aimed at preserving basic functions of tooth. Removing of part of the root or periradicular tissues, destroyed during the pathological process, and which cannot be treated, is expected;

3.surgical —removing of the affected tooth (extraction) and pathologically changed alveolar bone;

4.physical.

Conservative treatment of periodontitis is carried out with purpose to remove the source of periodontal ligament contamination (pathologically changed pulp tissues, dentin, microflora of root canal and dentinal tubules) with careful instrumentation, medical treatment of root canals and their obturation. Removal of the infection source should allow the inflammation of periodontal ligament to subside and symptoms to dissipate. These stimulate the regeneration of periodontal ligament and periradicular tissues.

Indications for conservative treatment:

1.Acute and chronic apical periodontitis of one-rooted teeth (with good negotiate canals) and absence of significant changes in periradicular tissues.

2.Acute and chronic apical periodontitis of multy-rooted teeth (with good negotiate palatinal canals of upper teeth and good distal canals of lower teeth) and absence of significant changes in periradicular tissues.

The main tasks of conservative treatment are:

1.Pain elimination.

2.Drainage of periodontal space.

3.Conduction of medicinal (antibacterial and anti-inflammatory) treatment of root canals.

4.Stopping further spread of inflammatory process in periradicular tissues.

5.Restoration of anatomical form and function of teeth.

Treatment of acute infectious apical periodontitis. The acute infectious apical periodontitis has very short phase of intoxication, but strongly expressed exudation. Exudate formed in periodontal space and periradicular tissues can spread into adjoining tissues in various ways: through a root canal, through the alveolar bone of jaw under periosteum from vestibular or lingual (palatal) side. The main goals of acute infectious apical periodontitis (both serous and purulent) treatment are: pain reducing, performing the drainage for releasing purulence (exudates), conducting antimicrobial and antibacterial treatment, stopping the spread of inflammation in

periradicular tissues, regeneration of periodontal ligament and periradicular tissues with restoration of tooth anatomic form and functions.

After cleaning the root canal the dentist reaches the tooth apex and apical foramen. Once the tooth is opened for treatment, drainage is carried out throughout the canal and remains open. Warm saline or antiseptic rinses are used for approximately 24-48 hours after the drainage procedure.

At secondary appointment (visit) such stages of medical treatment are carried out: thorough final preparation of carious cavity (tooth), root canal preparation, obturation of root canal, radiographic studies necessary for assessing the quality of root canal filling, placing temporary or permanent restoration (composites, amalgam, inlay, artificial crown) after the intraorifice barrier was placed.

Treatment of chronic apical periodontitis. The main goals of treatment are: elimination of the source of periodontal ligament contamination, further suppression of pathologic microflora of root canals and its diverse ramifications; providing conditions for regeneration of periradicular tissues; hyposensibilization of patient's organism.

The next goals of treatment are thoroughly preparation of the carious cavity, providing access to pulp chamber and root canals, root canal preparation. At this stage the preparation is fully performed, including canal negotiation, canal shaping, und, finally, apical preparation. After preparation is complete the medicaments such as calcium hydroxide (antiseptics, sorbents, enzymes, etc.) are placed with a long cotton pellet and temporary filling is placed.

At secondary appointment. There are obturation of root canal, radiographic studies necessary for assessing the quality of root canal filling, placing temporary or permanent restoration (composites, amalgam, inlay, artificial crown) after the intraorifice barrier was placed.

Conservative-surgical methods of apical periodontitis treatment

Several factors have resulted in a significant impact on the indications for and the application of endodontic surgery. Even though the success rate of nonsurgical endodontic treatments is high, failures do occur. There is a few absolute contraindications to endodontic surgery. Most contraindications are relative, and they are usually limited to three areas: (1) the patient's medical status, (2) anatomic considerations, and (3) the dentist's skills and experience.

Surgical drainage is indicated for purulent and/or hemorrhagic exudate forms; within the soft tissue or alveolar bone as a result of symptomatic periradicular abscess. A significant reduction of pain and a decrease in the disease duration will follow the pressure reduction and the elimination of by-products of inflammation and infection. Surgical drainage may be accomplished by (1) incision and drainage (I & D) of the soft tissue or (2) trephination of the alveolar cortical plate.

Endodontic surgery encompasses surgical procedures performed to remove the causative agents of periradicular pathosis and to restore the periodontium to a state of biologic and functional health. These procedures may be classified as follows (the most frequently used):

•Root-end resection.

•Root resection (amputation).

•Hemisection.

Conservative treatment is preferable in periodontitis treatment in permanent teeth in children. There are several indications for permanent teeth extraction: — the teeth are a source of acute odontogenous osteomyelitis; — technical impossibility for conservative or conservative-

operative treatment of periodontitis; — significant destruction of a crown tooth part, if its root cannot be used for prosthetics; — irreciprocal complications in the tooth treatment (perforation of the pulp chamber bottom or a root in the exacerbation stage) in case of impossible operative-conservative treatment. Treatment of acute infectious periodontitis and exacerbation of chronic periodontitis in mature permanent teeth. Conservative treatment of acute infectious periodontitis is aimed at inflammation liquidation in periodontium, pain relief and prevention of the inflammatory process diffusion to other regions of the maxillofacial area. Due to presence of serous or purulent exudate it is necessary to provide its outflow in the least traumatic way — through the root canal — by removal of the necrotic masses from it. If the exudate outflow is not provided, it is necessary to open the apical foramen. As a rule, acute infectious periodontitis and exacerbation of chronic periodontitis of permanent teeth are treated in several visits. The first visit suppose the following manipulations: — anesthesia; — pulp chamber opening with the use of a high-speed handpiece; at this stage it is advisable to deduce the tooth from the occlusion by partial grinding of the cutting edge or cusps. It will prevent the tooth from splitting at biting before it is final restoration; — putrid mass removal from the canal using antiseptic solution and root canal files of appropriate sizes; — opening of an apical foramen with a thin file or a reamer (if there is no exudate outflow through the root canal); — root canal instrumentation: removal of the infected predentine layer from the canal walls followed by a medication with an antiseptic solution; — in case of subgingival or subperiostal abscess — its opening and drainage (performed in a surgery). The subsequent doctor's tactics in the first visit depends 011 the clinics. Two methods of the tooth treatment are possible — «open» and «closed». The «open» method leaves the tooth open after the primary canal instrumentation and medication. This method is applied in case of plentiful purulent exudation from the canal. The following treatment is prescribed in this case: — frequent mouth rinsing with 0,5 % sodium hydrocarbonate solution (approximately 1/4 teaspoons of baking soda per glass of warm water); — drink a lot of liquid; consume non-irritating food which does not require intensive chewing; — ensure drainage preservation through the root canal (the tooth should be open; for the meal time it may be closed with a cotton ball); — a course of antibiotics, hyposensitizing drugs, non-narcotic analgesics in an age dose is prescribed in case of expressed inflammatory reaction, fervescence, child's general weakness. The second visit should be appointed in a day. The doctor estimates the following: patient's general condition; presence of changes of the mucosa in the causative tooth root projection; pain symptoms at its palpation; tooth sensitivity at percussion; presence and character of the root canal exudation. As a rule, in case of correct manipulations performed in the first visit, the acute inflammatory features remit within a day; so the temporary canal obturation with a medicamental substance becomes possible. The next visit includes the following procedures: — final root canal instrumentation: complete removal of infected pre-dentine from the walls; formation of the canal; plentiful and careful antiseptics irrigation (sodium hypochlorite, chlorhexidin, etc.); — drying of the canal with cotton turundas and paper pins; applying therapeutic substance with antiseptic and anti-inflammatory drugs on a turunda or (more preferably) in a pasta form into the root canal. Pastes with calcium hydroxide in high concentrations (pH more than 12), antibiotics, corticosteroids, metronidazole, and iodoform pastes may be used for these purposes. — hermetic bandage from a temporary filling material (dentine- pastes, water dentine, etc.). The «closed» treatment method of acute or exacerbated periodontitis supposes a complete root canal instrumentation and its temporary obturaion by a therapeutic substance in the same visit. The method is used in case of the exudate absence (or its presence in a small amount) after the apical foramen disclosure, as well as at the stage of serous inflammation in periodontium. The «closed» method requires an extremely careful canal instrumentation and medication: complete removal of the infected dentine, final canal formation, plentiful and durable canal irrigation with a sodium hypochlorite solution, application of other antiseptics. The processed and dried canal should be filled with a high-concentration paste with calcium hydroxide, water-based preferably, with pH not

less than 12-12.5. In case of expressed inflammation drugs with anti-inflammatory and antibacterial properties (containing antibiotics and glucocorticoids) can be used. However, the admissible terms of application of pastes containing corticosteroid hormones should not be exceeded. A long-term application of a hormonal drugs can slow down regeneration processes in periapical tissues, using «closed» method the patient is practically always prescribed a general treatment: nonsteroid antiinflammatory drugs, antibiotics, antihistaminics, a fortifying therapy (vitamins), and plentiful consumption liquid. The treatment termination regardless the method used depends on the optimal duration of the use of medicinal drugs for the root bandage (usually it makes 1—7 days). The root canal can be filled at the following conditions: — absence of spontaneous pain in the tooth; — absence of facial asymmetry, mucosa edema, submucosal or subperiostal abscess; — painless tooth percussion; — painless palpation of gingiva and mucosa fold in the causative tooth area; — absence of exudate in the canal (checked by absence of color change of an inserted turunda with iodonolum); — absence of a unpleasant smell in the canal; — light dentinal sawdust from the canal walls in case of their slight instrumentation with an Hfile. If any of the abovementioned conditions is absent, the doctor should canal instrumentation and medication and its temporary obturation with the use of therapeutic pastes or liquids on turundas. The last visit suppose the following manipulations: — removal of the hermetic bandage; — careful instrumentation of the root canal; drying; — permanent canal obturation by any method with the use of gutta-percha and sealers; — radiological control of the canal obturation quality; tooth crown restoration with permanent filling materials ora standard crown (the final restoration may be postponed until the next visit, as it may require a significant amount of time). Treatment of acute toxic periodontitis in permanent teeth In most case toxic periodontitis in permanent teeth (same as in deciduous teeth) results from the use of an arsenic paste or a phenol group antiseptics. Treatment is aimed at neutralization or elimination of a toxic sub-stance and the inflammation liquidation in periodontium. As a rule the developing process has a serous character and it is accompanied by an expressed inflammatory reaction. The first visit envisages the following manipulations: — anesthesia; — pulp chamber disclosure with the use of a high-speed handpiece; — removal of devitalized pulp (in case of arsenic periodontitis) or turundas with an irritating therapeutic substance; — complete root canal instrumentation; — processing of the root canal with an antidote preparation: in case ol arsenic periodontitis - 5% unithiolum solution, sodium thiosulfurieum, 5% iodine solution, 1% iodinolum solution; in case of tissue damages by phenol - castor oil or 10% anesthesinum emulsion in castor oil; — applying a turunda with an antidote into the root canal; — hermetic filling for 24 hours. The tooth should not be left open because of possible infection penetration into periodontium through the canal. Prescriptions: — analgesics internally in case of expressed pain reaction. The child is appointed for daily visits. In case of continued pain and painful percussion the canal medication with an antidote should be repeated and the intracanal bandage should be renewed for another day. In this case some intracanal drugs with antiinflamatory action can also be used (Ledermix, Fokalmin, and Pulmoseptin). The root canal should be finally obturated after all the acute periodontitis symptoms disappear. Treatment of an acute traumatic periodontitis in permanent teeth. Most often periodontal traumas result from exo-apical protrusion of endodontic tools. Thus, as a rule, the traumatic factor is accompanied with the infectious one. «The closed» method (according to the acute infectious periodontitis treatment scheme) is recommended for the tooth treatment. Preparation with antibacterial and anti-inflammatory action should be used for the temporary obturation. Treatment of acute periodontitis caused by combined mechanical and chemical damages of periodontium (due to the filling material protrusion over the apical foramen) or by a hematoma formation in periodontium (as a result of a traumatic pulp extirpation), is mainly carried out by physiotherapeutic methods. Prescription: UHF therapy or microwave therapy (5—6 sessions); fluctuorization with a single-step electrophoresis of 10% calcium chloric! solution (in case of expressed pain syndrome); mouth rinsing with 0.5-1%- sodium hydrocarbonate solution; analgesics internally. In case of acute

purulent periodontitis development as a result of a hematoma becoming infected, the filling material should be removed from the canal. Further treatment is carried out according to the treatment scheme for acute periodontitis of infectious origin. Treatment of chronic periodontitis in permanent teeth with mature roots. Treatment of chronic periodontitis in permanent teeth (as well as in deciduous teeth) can be performed in one or several visits. One-visit treatment is possible under the following conditions: — absence of gangrenous tissues with putrefactive smell in the canal: — absence of granulation grown into the canal; — absence of aggravations in the history: — technical possibility to perform the complete root canal instrumentation and medication and achieve its complete dryness in a single visit; — good general health condition of the child; — the child does not take antibiotics, corticosteroid drugs, cytostatics and other immunodepressive drugs. If all of these conditionsare present, the doctor performs a complete root canals instrumentation and medication with next permanent canals obturation followed by the crown part restoration. However, if the root canal passage is complicated, the child is impatient or weakened, the number of visits should be increased. In the first visit the following manipulation are performed: — necretomy and the carious cavity formation; — pulp chamber disclosure with the use of a high-speed handpiece; — putrid mass removal from the canal using an antiseptic solution with root canal files of appropriate sizes; — in the presence of granulations ingrown into the canal, they should be removed either with the use of injection anesthesia, or by shortterm (up to 5 minutes) processing of the granulations with camphorphenol, camphoroparamonochlorphenol, a mixture of phenol and monochlorphenol with anesthesinum, inserted into the canal in a turunda. The procedure should be performed with special care, there is a danger of the oral cavity mucosa chemical burn;

**TOPIC 54.**

**MODULE 3**

**TOPIC 55.**

## DIFFERENTIAL DIAGNOSIS OF CATARRHAL AND HYPERTROPHIC GINGIVITIS. TREATMENT AND PREVENTION. THE USE OF PHYSICAL FACTORS IN DIAGNOSIS AND TREATMENT.

Gingivitis is the inflammation of gums, conditioned by the unfavorable action of local and general factors, that flows without violation of integrity of tooth-gingival connection. Catarrhal gingivitis. Patients grumble about the unpleasant feeling in gums, feeling of itch. Odor nuisance from a mouth, by curvature of taste, sanguifluousness of gums during a meal or cleaning of teeth. The general of patients suffers small, however the periods of intensifying can be accompanied by indisposition, lowgrade temperature.

Acute catarrhal gingivitis is characteristic for the period of eruption and changes of teeth. It is observed at acute infectious and other inner diseases.

Chronic catarrhal gingivitis differs in the protracted languid motion; complaints are poorly expressed. At an objective inspection mark an edema, hyperemia, cyanosys clear, a mechanical irritation is accompanied by bleeding. On teeth is enhanceable maintenance of soft dental raid(patients avoid to clean teeth as a result of the pain feeling and sanguifluousness of gums). The amount of gingival liquid increases, the positive test of Schiller is determined also - Писарева, increase emigration of leucocytes for Yasinivsky; mionectic firmness of capillaries is at a vacuum test for Kulagenko and other It follows remember, that catarrhal gingivitis can be the symptom of general disease, the early exposure of that has certain curatively, is a prophylactic value.

Treatment of catarrhal gingivitis includes: - removal of local irritating factors(moving away of dental sedimentations, stopping of carious cavities, first of all II, V of class for Blek, moving away of the unrationally made prosthetic appliances of т. д.); - local, and in case of necessity and general ethiotropic (antimicrobial) therapy; - local use of antiinflammatory facilities; - increase of local heterospecific resistance; - physical therapy methods. Preparations of choice for empiric etiotropic therapy are ethonij, chlorhexidine, «Paragel», «Metrogyl-Denta», Hinoxidin, Dioxidin as preparations of choice for empiric etiotropic therapy. For antiinflammatory therapy of generalized of catarrhal gingivitis recommend to use non steroid antiinflammatory facilities: to the mephenaminat natrium, nimesulid, movalis, indomethacin, Ibufrofenum, diclofenac and other. Them it follows to use in the holiatry of catarrhal gingivitis only in that case, when rational local interference, antibacterial and imunomodulatine therapy not able to stop an inflammatory process at gums. For general tonic treatment appoint vitamins And, C, E, ascorutin, polivitamins in combination with microelements – triovit, duovit, revit and other Physiotherapy. For fixing of results of medicamental therapy it is necessary to conduct the row of physical therapy events : auto-, hydro-, vibratory massage, hydropathy, electrophoresis, photoradiotherapy and other metods.

Hypertrophic gingivitis

This is a chronic inflammatory process in the gums, accompanied by proliferative phenomena with the growth of fibrous structures of the lamina propria of the gingival mucosa and the proliferation of the basal layer of the epithelium. It develops very slowly, exacerbations and remissions are possible.

Among the etiological factors are local irritants, as well as violations of some systems of the body, especially endocrine. Hypertrophic gingivitis often occurs during puberty and in women

during pregnancy. It can occur with hypovitaminosis c, metabolic disorders, in particular carbohydrates. It is important to survive certain drugs ( diphenine , diphenyl-hydantoin , which are against convulsive drugs).

There are localized and generalized forms. The localized form is caused by the action of local damaging factors, the generalized form is observed in concomitant diseases. According to the degree of severity, there are I, II, III degree depending on the degree of hypertrophy.

I degree – gingiva covers the crown of 1/3

II degree – gingiva covers the crown of 1/2

III degree – gingiva covers the crown of 2/3

Hypertrophic gingivitis is localized mainly on the vestibular surface of the frontal gums. Lesions of the oral surface most often occur in blood diseases ( hyperplastic syndrome in leukemia). Particular attention should be paid to this, as it may be the only early clinical symptom of leukemia.

According to clinical and morphological features, there are 2 forms of the disease: edematous (inflammatory) and fibrous.

Edematous ( granulating ) form

Complaints:

Growth of the gums, unusual appearance of the gums, bad breath, bleeding and pain in the gums, which is aggravated by eating and chewing. May exacerbate.

Objectively:

Changes in the relief of the gums due to hyperplasia of the gingival papillae and roller-like thickening of the marginal part of the gums. The shape of the gingival papillae is incorrect, the appearance of the gums in the advanced stages is distorted.

The gingival papillae and gingival margin are hyperemic with pronounced cyanosis, the alveolar part of the gums is in a state of chronic catarrhal inflammation. Pronounced swelling and loose gums, they lag behind the necks of the teeth. Pain on palpation of the gums, bleeding on examination of the gingival pocket is determined.

Due to severe edema and hyperplasia formation of different depths false gingival pocket filled with serous fluid, but dentogingival of ' unity is not broken.

This form of GG has the peculiarities of the course in adolescents during puberty: it is characterized by a tendency to frequent exacerbations, rapid progression, excessive signs of inflammation and excessive hypertrophy of the gingival papillae.

Fibrous form

Complaints:

At insignificant hypertrophy of gums are absent, or on an unusual kind of gums. The disease develops slowly and imperceptibly for the patient, practically does not give exacerbations.

Objectively:

Changes in the relief of the gums due to hyperplasia of the gingival papillae and roller-like thickening of the gingival margin.

Gum papillae pale pink, close to the necks of the teeth. At inspection of a gingival furrow bleeding is not revealed. There is no exudate in the fake pockets. Hypertrophied areas of the gums on palpation are dense and painless. There may be hypertrophy of the gums not only from the vestibular but also from the oral surface of the teeth.

Data of additional methods of inspection at HG

The Shillev-Pisarev test is sharply positive at the edematous form.

Indicators of all hygienic indices indicate unsatisfactory hygienic condition of the oral cavity.

Formalin test ( Kotzchke 2 ) is negative (if there is no violation of the integrity of the gum epithelium due to injury).

Decrease in stability of capillaries at vacuum test according to Kulazhenko (10-20 sec).

There are no changes on the radiograph, with a long course of the disease, osteoporosis of the vertices of the interalveolar septa is possible.

Mandatory ' if omitted is a blood test to exclude displays of hyperplastic syndrome in diseases of the blood.

The amount of gingival fluid is sharply increased in the edematous form.

Pathomorphological changes in HG

In the granulating form - parakeratosis in the superficial cells of the epithelium, vacuolar dystrophy in the cells of the spiny layer. Proliferation of the basal layer of the epithelium. In its own plate - the growth of fibrous structures, round-cell leukocyte infiltrates. Granulation tissue is formed and grows. Capillary dilation, venous stasis.

In the fibrous form - a violation of the keratinization of the epithelium. In own plate of gums growth of dense collagen fibers. Lymphocytic infiltrates around blood vessels . The walls of blood vessels are thickened.

Differential diagnosis of HG

Intra - syndromic - between two forms, chronic and acute, severity.

with symptomatic HG in periodontitis ;

with symptomatic gingivitis on the background of somatic pathology (blood diseases, endocrine system, metabolic disorders);

with symptomatic gingivitis with hypovitaminosis C.

fibrous form - from fibromatosis of the gums, epulis and other tumor-like diseases.

| | Catarrhal gingivitis | Hypertrophic gingivitis |
|---|---|---|
| Etiology | Plaque microflora, infectious diseases in the past medical history, impact of the stressors, vitamin and microelement deficiency, internal | Endocrine diseases, pregnancy, puberty, menopause, blood diseases (leukemia), the use of certain |

| | | |
|---|---|---|
| | organ pathology, excessive alcohol consumption and smoking tobacco | drugs (diphenylamine, cyclosporine). |
| Color of the gums | Congestive hyperemia in chronic course, bright hyperemia in acute course | Congestive hyperemia in granulating form, absence in fibrous form |
| Gingival bleeding | When brushing teeth and eating solid foods | When brushing teeth in granulating form, absence in fibrous form |
| Periodontal pocket | Edema-related periodontal pocket | Hypertrophy-related periodontal pocket |
| General condition | General weakness, fever in acute condition | Normal |

Differential diagnosis of ulcerative-necrotic gingivitis. Treatment and prevention.

Ulcerative (ulcerative-necrotic) gingivitis is an inflammatory process in the mucous membrane gum, which is characterized by the predominance of an altered component, a violation of integrity

and tissue necrosis. It is observed less often than other forms of gingivitis.

In etiology, the leading role is played by fuzzy-peptidic symbiosis: fusobacterium (gram-negative anaerobic sticks) and spirochetes. Acute ulcerative-necrotic gingivitis develops like the rule, against the background of reducing the immunological reactivity of the organism as a result of the suffered acute respiratory viral diseases, infectious diseases of bacterial etiology, with acute herpetic stomatitis, as well as deficiency of ascorbic acid.

Development the disease contributes to reducing the local immunity of the oral cavity, the presence of local traumatic factors, lack of hygienic care of the oral cavity.

Ulcerative gingivitis usually has an acute course. Depending on the prevalence process and severity of general clinical manifestations distinguish between light, medium and severe səˈvi(ə)r degree of disease.

In the prodromal period (1-2 days), the patient has an increase in temperature body to 37-38 C, weakness, headache, loss of appetite, sleep disturbance.

At first signs of acute catarrhal gingivitis are observed: swelling, hyperemia and bleeding of the gum mucosa. Subsequently, against the background of pronounced catarrhal changes occurs necrosis of the gingival papilla and marginal edema.

Patient complains of severe pain and bleeding of the gums, which increase when eating food, rotten smell from the mouth, elevated salivation.

The onset of the disease is manifested by the lesion of the gum mucosa in the area 1-3 teeth that corresponds to a slight degree of gravity. On the surface of the gum on the background of edema and pronounced hyperemia is a dirty-gray necrotic plaque. After it is removed it's exposed sharply painful bleeding ulcerous surface. Ejaculatory papillae lose conical shape,

compacted

There is a significant amount of soft plaque, very unpleasant smell from the mouth cavity, increased salivation, as well as an increase in regional lymph nodes.

The average degree of gravity is characterized by the diffuse spread of the process along marginal gums, higher body temperature (from 37.5 to 39 C) and an increase in signs of intoxication. A patient may be affected by sleep, nausea and diarrhea. When progressing

the process of interdental papillae can be completely necrotic

The severe course of acute ulcerative-necrotic gingivitis is observed, as a rule, at weakened patients. Necrosis can spread to the alveolar part of the gum and other parts.

Sometimes the disease begins with lesions of retro-molar areas and palatine tonsils (angina Vensana).

The general condition of these patients is significantly disturbed due to an increase in body temperature

(more than 390C) and severe intoxication.

Duration of the period of pronounced clinical manifestations in acute ulcerative gingivitis is, on average, 7 to 10 days, depending on the severity of the disease, timeliness and quality of assistance.

X-ray changes in the periodontal tissues during the acute course of ulcer-necrotic gingivitis is absent.

Differential diagnosis of acute ulcerative gingivitis is carried out with necrotic changes in gum mucus in idiopathic diseases with progressive lysis periodontal tissues (hereditary neutropenia, histiocytosis), as well as in diseases of the blood (leukemia).

Treatment depends on the characteristics of pathogenesis, severity, the nature of the course process and age of the patient.

Local treatment:

1. Anesthesia (10% lidocaine gel, Cimistad gel or other anesthetics in the form of applications).

2. Removal of necrotized tissues (application of proteolytic enzymes - trypsin, chymotrypsin, terylitin 1-2 times a day, ointment "lingezin", which, in addition to the enzyme, contains also antibiotic; antiseptics - chlorhexidine, hexytidine).

3. After the removal of necrotic tissue, antibacterial therapy is performed (metronidazole or complex drugs containing chlorhexidine and metronidazole "Metrogil-denta"; at a severe course of the ulcerous process it is expedient use broad spectrum antibiotics - lincomycin, tetracycline, in the form of instillations or applications).

4. Anti-inflammatory therapy (NSAIDs - 0.1-0.2% aqueous solution of mefenamine sodium salt,

1% gel or jelly "Dickler", 1% emulgel "Voltaren", herbal means origin - flowers of chamomile, calendula, sage, nettle leaves, artificial lysozyme "Lisobact")

5. After cleaning the surface from necrotic and fibrinous plaque and after

The removal of acute inflammation requires the use of keratoplasty (oil solutions of vitamin A and E, multivitamin complex "Aevit", rosehip oil, caratolin, sea buckthorn oil, as well as biogenic stimulants: kalanchoe juice, jelly "Solkoseril", they are used in the form of applications).

6. Training of methods of individual care of the oral cavity, hygienic control for their implementation and recommendations on the choice of hygiene products.

7. Elimination of local factors contributing to the development of ulcerative-necrotic

gingivitis (performed after the completion of tissue epithelization).

General treatment with a mild severity of acute ulcerative gingivitis is in removing the phenomena of intoxication at home (repeated drinking of vitamin and enveloping liquid - apple-carrot juice, berry or milk kissel, mozz). Food should be be energetically complete, soft consistency (buckwheat, oatmeal porridge), protein foods that are easy digested (fish, poultry meat), limited use of refined carbohydrates.

Also prescribed orally complex vitamin preparations that contain ascorbic acid, vitamins A, P, E and B (Decamivet, Macrovit, Vitrum).

## TOPIC 56.

## DIFFERENTIAL DIAGNOSIS OF LOCALIZED PERIODONTITIS. THE USE OF PHYSICAL FACTORS IN DIAGNOSIS AND TREATMENT.

Localized periodontitis is a disease that characterized by inflammation and progressive destruction of periodontal tissues (gum, periodontal disease, interalveolar septum, cement root) in the site one or several teeth, and may result in loss damaged teeth.

Clinical characteristic of localized Periodontitis is presented as acquired symptomatic complexes: symptomatic gingivitis, periodontal pockets, destruction of root tooth cement, osteopathy, breaking of

Classification (MF Danilevsky 1994):

Form: catarrhal, hypertrophic, ulcerative ,atrophic

The course: acute, chronic

Depth of defeat: soft tissues and alveolar bone

Degree of development: initial, I degree, II degree, III degree.

Prevalence of the process: focal

Initial degree of localized periodontitis depends on the nature of the course of symptomatic gingivitis (acute or chronic). Clinically determined symptomatic light gingivitis (papillitis), dental deposits, gums pockets, traumatic occlusion.

X-ray defining a violation of a compact plate of vertebrae of alveolar partitions (destruction), insignificant resorption of vertices of partitions and expansion , periodontological cracks in the cranial region.

At I stage localized periodontitis is clinically is defined symptomatic gingivitis, periodontal pockets up to 4 mm deep with moderate serous-purulent content, abnormal tooth movement and degree, expressed traumatic occlusion. Radiologically determined resorption of interalveolar partitions to 1/3 length root, extension of the periodontance gap.

For II degree of localized periodontitis characteristic symptomatic gingivitis of the lung or moderate severity (marginal gingivitis), periodontal pockets up to 6 mm with serous-purulent contents, traumatic occlusion, movement of teeth I-II stages. Radiologically determined expansion periodontance gap, resorption of interalveolar partitions up to 1/2 of the length of the root. Often availability caries of cement root.

With localized periodontitis III degree the severity of clinical manifestations is reduced to severe symptomatic gingivitis, severe traumatic occlusion, depression of periodontal pockets up to 6-8 mm, tooth movement of II-III stages. On the X-ray - resorption of bone tissue is determined by 2/3 and more length of the root, extension of the periodontance gap, decalcification of root tooth cement

Chronic localized periodontitis is characterized by chronic symptomatic gingivitis (catarrhal, hypertrophic) with moderate content serous or serous-purulent periodontal exudates pockets

- Surgical methods: curettage - at depth periodontal pockets 3-5 mm; gingivotomy - at depth of pockets more than 5 mm.( II – III degree)

| | Localized periodontitis |
|---|---|

| | |
|---|---|
| Etiology | Mechanical trauma (inappropriate preparation and fixation of the fixed dentures), occlusal trauma (malocclusion, premature removal of molars), etc. |
| Color of the gums | Congestive hyperemia in chronic course, bright hyperemia in acute course |
| Gingival bleeding | Minor bleeding and tender palpable |
| Dental calculus deposits | Supra- and sub-gingival |
| Tooth roots baring | Baring according to the severity |
| Periodontal pocket | Periodontal pocket of various depth |
| Teeth mobility | Class I-III pathological mobility |
| The state of regional lymphatic nodes | Enlarged, tender palpable in the acute course |
| Findings of additional studies | Radiography shows destruction of the compact plate, resorption of the interalveolar septa on the bounded area of the jaw |

Differential diagnosis of periodontitis. Modern methods of treatment and prevention

Symptom complex in periodontitis :

- symptomatic gingivitis

- periodontal pocket

- under ' gingival tartar

- traumatic occlusion

- progressive resorption of alveolar bone.

Symptomatic gingivitis.

It most often manifests itself in the form of catarrhal gingivitis - in about 70% of patients. Symptomatic catarrhal gingivitis may be chronic or acute.

If the patient develops symptomatic hypertrophic gingivitis - there is a granulating (edematous) form. The course is often chronic, but possible exacerbation, in which there are more pronounced signs of gingivitis.

At development of symptomatic ulcerative gingivitis its chronic course is most often observed, thus insignificant necrosis of tops of gingival papillae or marginal edge of gums is noted. Necrotic surfaces are separated from the surrounding gingival mucosa by a demarcation line in the form of a hyperemic strip. Sometimes there is an acute course with more pronounced necrotic phenomena and the formation of deep periodontal pockets with serous-purulent or purulent exudate.

Periodontal pocket. Are required ' necessarily a sign of periodontitis .

In the initial stages of periodontitis allegedly preserved the integrity of periodontal of ' unity, however histopathological studies reveal a violation of his integrity. In the clinic, this can be confirmed by formalin testing Parma ( Kotzchke 2) .

Further due to destruction of circular called ' bandages dental cement between the tooth and gums inner surface formed recesses of different depths - periodontal pocket. Their depth varies from 2 to 10 mm . As a result of the destruction of the ' unity of the epithelial attachment cuticle lost tooth mechanisms that restrain proliferation and maturation of immature epithelial attachment. As a result, the epithelium grows into the depth of the connective tissue of the gums and its differentiation into the multilayered squamous epithelium of the gums. At the bottom of the periodontal pocket proliferates granulation tissue. The periodontal pocket is filled with exudate - serous, serous-purulent or purulent.

Thus, the walls of the periodontal pocket: tooth cementum, ingrown multilayered gum epithelium, granulation tissue at the bottom.

Content periodontal pockets, fluid, under ' gingival tartar, dental plaque.

Traumatic occlusion.

The primary traumatic occlusion - develops against the background of an intact periodontal due to excessively second -largest or the usual trend for chewing pressure.

Secondary traumatic occlusion develops against the background of periodontal disease, when even the usual occlusal load exceeds the compensatory capacity of the periodontium, becoming a traumatic factor. Due to the mobility during the action of masticatory pressure, the teeth are deflected, as a result of which their physiological abrasion does not occur. Thus, teeth with intact periodontium are erased, while in areas with affected periodontium, the teeth retain bumps on the masticatory surface and the cutting edge. Thus, these bumps when closing the dentition are the first to come into contact with the teeth-antagonists, which causes traumatic overload of such teeth and leads to the formation of so-called traumatic nodes.

The mobility of the teeth leads to the displacement of the teeth vestibularly or orally and their extension from the holes. The movements of the lower jaw change, because the patient reflexively protects the moving or displaced teeth from the action of masticatory pressure. This leads to disruption of chewing tone ' muscles and changes in the temporomandibular joint.

The movement of teeth leads to the loss of interdental contacts and violation of the integrity of the dentition. The dentition ceases to function as a single system.

The degree of mobility of the teeth does not always correspond to the severity of periodontitis . At a chronic current - degree of mobility, as a rule, coincides with degree of severity of a disease (I degree of mobility at I degree of severity, etc.). In the acute course of generalized periodontitis, the degree of mobility is always higher than the degree of severity (in the first degree of severity of periodontitis, mobility can reach II or even III degree).

Progressive resorption of alveolar bone.

The main radiological symptoms of periodontitis :

destruction of the vertices of the interalveolar septa

destruction of the cortical plate of the interalveolar septa

decrease in the height of the interalveolar septa

uneven resorption of interalveolar septa in different departments

vertical, horizontal and lacunar resorption

osteoporosis of the bone of the alveolar sprout

large-loop pattern of bone tissue

widening of the periodontal gap, especially in the marginal parts

formation of bone pockets.

The severity of periodontitis is set solely on the radiograph:

at the initial stage - destruction of the cortical plate of the interalveolar septa and a slight decrease in their height

at the I degree - decrease in height of interalveolar septa to 1/3 of their height

at the II degree - decrease in height of interalveolar septa within 1 / 3-2 / 3 of their height

at the III degree - decrease in height of interalveolar septa by 2/3 of their height and more.

Differences in chronic and acute periodontitis .

Chronic course.

It is characterized by the development of chronic symptomatic gingivitis with scanty clinical symptoms. In periodontal pockets scanty serous or serous-purulent exudate. The mobility of the teeth usually corresponds to the degree of bone resorption. The general condition of the body is unchanged. Radiologically - resorption of alveolar bone is more uniform, without formation of deep bone pockets. The phenomena of osteoporosis are weakly expressed.

Exacerbation of periodontitis .

Occurs when the body's reactivity decreases (for example, against the background of comorbidities). Clinically there is an exacerbation of symptomatic gingivitis, accompanied by severe pain, edema, redness, bleeding. The number of secretions from the periodontal pocket increases , they are purulent in nature. Sometimes on the background of the formation of deep pockets in the bone ' which tissues are formed of single or multiple periodontal abscesses. With ' are symptoms of intoxication (fever, headache, malaise, etc.). Tooth mobility is higher than the severity of the disease. Radiologically : uneven progressive resorption of alveolar bone, formation of bone pockets, severe osteoporosis of the bone. The periodontal fissure is expanded for a considerable extent.

Laboratory indicators.

Capillaroscopy and biomicroscopy . At a chronic course there is a desolation of capillaries, turbidity and cyanoticity of a capillary background, increase in number of functioning capillaries, venous stagnation, expansion of capillary loops, slowing down of a blood-groove. In the course of exacerbations than specified, there is excessive povnokriv ' I capillaries.

Vacuum test for Kulazhenko . Normally, a hematoma is formed in about 30-50 seconds. With generalized periodontitis, this time is reduced to 7-10 seconds in chronic and up to 3-5 seconds - in acute.

Migration of leukocytes into the oral cavity by the method of Yasinovsky . Norm - leukocytes up to 150 in 1 ml of flushing fluid, at least 80% of them are alive, the number of squamous epithelium does not exceed 50. In chronic periodontitis, the number of leukocytes doubles, the percentage of living neutrophils is below normal. The number of desquamated epithelial cells increases to 100. With exacerbation, the number of leukocytes increases 4 times, the percentage of living neutrophils is below normal. The number of squamous epithelial cells reaches 150.

Cytological examination. In chronic periodontitis, the content of active phagocytes is sharply reduced. With an acute course, the percentage of active neutrophilic granulocytes increases, which can be explained by a certain increase in the protective properties of periodontal tissues.

Microbiological research. In the case of chronic periodontitis , cocci, spindle-shaped rods, spirochetes, yeast-like fungi, and protozoa are found in periodontal pockets . In the acute course, the number of microorganisms detected by microscopic or bacteriological studies increases by 5-10 times, dominated by cocci or rod-shaped microorganisms.

Protein fractions of blood. In patients with chronic and acute GP there is a decrease in albumin, an increase in globulins, especially γ -globulins.

Phagocytic activity of leukocytes. Normally, the percentage of phagocytic neutrophils is 83-86%, and the phagocytic number is 5-6. These indicators in the chronic course of GP are at the lower limit of normal, in the acute course - at the upper limit.

Clinical course.

Initial degree.

Complaints of bleeding gums when chewing, brushing teeth; discomfort in the gums, itching and paresthesia of the gums. Sometimes there are no complaints, the pathological process without subjects ' of objective signs.

Anamnesis. It is quite difficult to find out when the disease started. As a rule, pain and bleeding bother the patient for several years, periodically increase and decrease.

Objectively . Local traumatic factors. Symptomatic catarrhal or hypertrophic gingivitis. Primary traumatic occlusion in places of low-quality prostheses, fillings, etc. Periodontal pocket 1-2 mm deep . Massive deposits of ' gingival tartar (especially on the lingual surfaces of the lower front teeth and upper molars vestibular surface - that is, in places of ductless glands). The deposits at ' gingival plaque in the interdental spaces and periodontal pockets. The necks of the teeth are not exposed. The mobility of the teeth is physiological. Positive formalin test.

Radiologically . Osteoporosis of the upper and middle third of the interalveolar septa, destruction and violation of the integrity of the compact plate, a slight expansion of the periodontal gap near the tops of the interalveolar septa.

I degree.

Complaints differ in chronic and acute course: in chronic - bleeding gums when brushing teeth, bad breath; with acute - severe pain, significant bleeding gums, hyperesthesia of the hard tissues of the teeth in the neck.

Clinical signs. Symptomatic gingivitis of diffuse nature. Hyperemia of the gums with a cyanotic tinge in the chronic course, bright redness - with acute. Decrease in height of tops of gingival papillae owing to what interdental spaces "gape". Exposure of the necks of the teeth,

resulting in the development of hyperesthesia of the hard tissues of the teeth. Periodontal pockets 2-3 mm deep , the content - serous-purulent, its number depends on the course. Massive deposits of ' gingival tartar, to a lesser extent - under ' gum . Expressed mobility of the lower front teeth (I degree - in chronic, II-III - in acute). Displacement of individual teeth, mainly in the frontal part of the mandible, secondary traumatic occlusion.

Radiologically . Complete destruction of the cortical layer at the apex and partially in the lateral areas of the interalveolar septa. Osteoporosis of the spongy substance of the interalveolar membrane within the middle third. Resorption of interalveolar septa up to 1/3 of their height (root length). Expansion of the periodontal gap within the upper third of the interalveolar septa.

II degree.

Complaints. In the chronic course of the process - scanty symptoms - bad breath, slight bleeding and sore gums, displacement and mobility of the teeth, the appearance of gaps between the teeth. At an exacerbation pain and bleeding are considerably expressed, spontaneous bleedings are possible.

Clinical signs. Symptomatic diffuse gingivitis with a pronounced decrease in the height of the gingival papillae and the "gaping" of the interdental spaces. Periodontal pockets 3-5 mm , filled with serous-purulent exudate, granulation tissue grows at the bottom. Massive postponement of over- and under ' gingival tartar. The necks and roots of the teeth are exposed by 2-3 mm , elongation of the clinical crown of the tooth. Hard tissue hyperesthesia . The mobility of the teeth (I-II degree in chronic course, II-III - in acute), is most pronounced in the area of the front teeth. Fan-shaped difference of frontal teeth.

With an acute course and the formation of a bone pocket, the formation of a periodontal abscess is possible . Its development is accompanied by almost constant pain in the affected teeth, sharp pain when biting the tooth, redness and swelling of the gums in this area. The general condition may be disturbed, body temperature rises.

Radiologically . Resorption of interalveolar septa within 1/3 to 2/3 of the tooth root length. Destruction of the cortical plate. Significant expansion of the periodontal gap in the middle third of the interalveolar septa. Diffuse osteoporosis of the interalveolar septa. Bone pockets may form near teeth with overhanging fillings.

III degree.

Complaints. In the chronic course, they are reduced to bad breath and significant mobility of the teeth, which causes an aesthetic defect and makes it difficult to eat. With exacerbation - severe pain and bleeding gums, possible deterioration of the general condition (due to intoxication of the body due to significant purulent discharge from periodontal pockets), possible enlargement of lymph nodes.

Anamnesis. Loss or removal of teeth due to their pathological mobility.

Clinical signs. Diffuse symptomatic gingivitis. The gums are congestively hyperemic , bleeding when touched. Periodontal pockets 8 mm or more deep , sometimes reaching the apex of the root. Their content is serous-purulent or purulent, there is a massive growth of granulation tissue in them. Massive postponement of over- and under ' gingival tartar. The necks of the teeth are exposed by 5-8 mm , the elongation of the clinical crown of the teeth. Hard tissue hyperesthesia . Mobility of teeth of the II-III degree. Displacement of teeth in different

directions. Defects of dentitions due to removal of movable teeth. The development of single or multiple periodontal abscesses is possible.

Radiologically . Absorption of alveolar bone reaches 2/3 of the length of the root and more. The cortical plate is destroyed almost throughout. The periodontal fissure is dilated throughout. Osteoporosis of the bone of the interalveolar membrane, which is still preserved. Bone pockets reaching the apex of the root are often identified.

Stabilization of the process appears after a comprehensive treatment. The patient has no complaints. Clear pale pink, tightly covering the roots of the teeth. The roots are exposed depending on the degree of periodontitis . There is no discharge from periodontal pockets and bleeding gums. No dental plaque. Teeth are stable or splinted with permanent splinting structures. These are manifestations of clinical stabilization (remission). Hygienic indicators within the norm. Pisarev-Schiller test is negative. RI is approaching 0. Kulazhenko's test within 50-60 seconds. Clinical and radiological stabilization in addition to clinical well-being characterized by the absence of foci of osteoporosis in , sealing spongy substance (osteosclerosis) of alveolar bone .

**Differential diagnosis of periodontitis**

Leukemia . P ri periodontitis with symptomatic hypertrophic and ulcerative gingivitis lesions gum expressed more of vestibular side, and with leukemia observed diffuse lesion on a background of pale mucous membrane, no bleeding, intoxication, temperature reactions, general weakness, no significant quantitative and qualitative changes in the blood .

Neutropenia ( cyclic and permanent) is characterized by constant infectious processes: boils on the skin, abscesses in the lungs, ulcers and perforations in the intestine. Inflammation in the periodontium , formation of gingival and periodontal pockets, resorption of alveolar bone, mobility of teeth. The pathological process is manifested in the first days of life, the process recurs ; up to 20 years the severity of the processes decreases. There may be eosinophilia and monocytosis.

Agranulocytosis is characterized by signs of acute sepsis and necrosis. There are necrosis of the mucous membrane and agranulocytic angina. Necrosis can begin with damage to the gums. The lesion occurs on the background of pale gums. There is severe mobility b teeth. Reminds periodontal etc. , but no hnoyevydilennya the presence ZYAK and necrotic phenomena. Absence or decrease in granulocytes in blood at decrease in number of all leukocytes.

Periodontal syndrome in diabetes. Characterized swollen, bright red with c and anotic hue desquamed gingival margin, which bleeds easily. ZYAK with and significant exudation and explosion of granulations, significant mobility of teeth and their movement along the vertical axis. Radiologically - lacunar type of resorption of alveolar bone.

Periodontal syndrome in Itsenko-Cushing's disease . Occurs as a result of primary damage to the pituitary gland with involvement of the adrenal glands , gonads and pancreas. There is obesity, subcutaneous hemorrhage, dysfunction of the gonads, pituitary diabetes, mental disorders. Clear sharply hyperemic , swollen, hemorrhages in them; mobility and displacement of teeth, growth of gingival papillae, periodontal pockets with purulent contents. X-rays reveal foci of osteoporosis and destruction of the alveolar sprout.

Disease Syndrome - a disease of chromosomal origin, type of mental retardation. The patient has trisomy on chromosome 21. In the oral cavity macroglossitis , folded tongue, microdontia . The mouth is constantly open, the mucous membrane dries out, cracks appear. Gingivitis

and generalized periodontitis develop , which clinically and radiologically manifests itself identically to periodontitis .

Parodontosis

Is a dystrophic periodontal disease, is quite rare (5-8% of dystrophic-inflammatory periodontal diseases). It begins imperceptibly, develops and progresses slowly, lasts for years.

Characteristic features:

symptomatic atrophic gingivitis

exposure of necks and roots of teeth

formation of wedge-shaped defects

moderate traumatic occlusion

lack of periodontal pockets

pathological mobility does not develop for a long time, it is manifested only in the late (II-III) stages of the disease.

The main radiological symptoms of parodontitis :

osteosclerosis of the bone of the alveolar sprout

fine-loop drawing of bone tissue

uniform decrease in the height of the interalveolar septa (horizontal resorption)

sclerosis of the tooth cavity, formation of denticles , petrifications

hypercementosis near the tops of the roots

cortical plate is stored for a long time

Initial degree.

Complaints. Has scanty clinical symptoms: some patients complain of itching, numbness in the gums, hyperesthesia of the hard tissues of the teeth.

Objectively . The mucous membrane of the gums is pale, there is no normal luster. Blunting of the tops of individual gingival papillae, most often in the area of the front teeth. The gums are dense, painless, do not bleed on examination. A slight (up to 1 mm ) exposure of the necks of the teeth, but the violation of the integrity of periodontal of ' unity is not observed. Hypersensitivity of hard tissues to all types of stimuli (chemical, temperature, mechanical). Minor traumatic occlusion. Tooth mobility is not observed . Pisarev-Schiller test is negative.

Radiologically . Areas of osteoporosis are preceded by areas of osteosclerosis. Uniform decrease in the height of the interalveolar septa to ¼ their height. Periodontal fissure without changes. The cortical plate is preserved throughout.

I degree.

Complaints. More pronounced signs of itching, a feeling of aches in the gums, hyperesthesia of the hard tissues of the teeth.

Objectively. Clear pale, dull, dense and painless. Exposure of necks on 1 - 1,5 mm . Gaps in the interdental spaces. Formation of shallow wedge-shaped defects on the front teeth. Traumatic

occlusion, overload of individual teeth. Tooth mobility is not observed. Pisarev-Schiller test is negative.

Radiologically . Osteosclerosis of the alveolar bone. Uniform decrease in the height of the interalveolar septa to 1/3 of their height. Periodontal fissure without changes. The cortical plate is preserved throughout.

II degree.

Complaints are similar.

Objectively. Clear pale, dull, dense and painless. Exposure of tooth roots up to 5-6 mm . The interdental spaces gape. Development of wedge-shaped defects on the vestibular surface of the teeth. Hyperesthesia of the hard tissues of the tooth. Slight delay on ' gingival tartar and plaque pigmented. Traumatic occlusion, overload of individual teeth. Tooth mobility can be manifested in the first degree. Pisarev-Schiller test is negative.

Radiologically . Osteosclerosis of the alveolar bone. Uniform decrease in the height of the interalveolar septa to 2/3 of their height. Periodontal fissure without changes. The cortical plate is preserved throughout.

III degree.

Complaints of hypersensitivity of the exposed necks of the teeth. Displacement and divergence of teeth.

Objectively. The gums are dense, anemic, do not bleed - atrophic gingivitis is most pronounced. Slight delay on ' gingival plaque and pigmented coating. The necks of the teeth can be exposed by 5-8 mm . Pathological mobility is either absent at all, or there is mobility of I-II degree. Fan-shaped divergence of front teeth is possible. Wedge-shaped defects develop on the vestibular surfaces of the teeth. Pisarev-Schiller test is negative.

Radiologically . Osteosclerosis of the alveolar bone. Uniform decrease in the height of the interalveolar septa by 2/3 of their height and more. Periodontal fissure without changes. The cortical plate is preserved throughout.

Differential diagnosis is performed with atrophic gingivitis, premature and senile periodontal atrophy.

## TOPIC 57.

## DIFFERENTIAL DIAGNOSIS OF PERIODONTAL DISEASE. MODERN METHODS OF TREATMENT AND PREVENTION. THE USE OF PHYSICAL FACTORS IN THE DIAGNOSIS AND TREATMENT OF DYSTROPHIC-INFLAMMATORY AND DYSTROPHIC PERIODONTAL DISEASES.

Diagnosis of periodontal diseases

The information regarding the condition of the various periodontal structures (the gingiva, the periodontal ligament, the root cementum and the alveolar bone) which has been obtained through the comprehensive examination presented above should form the basis for a proper assessment of the periodontal condition. It is advantageous to give each tooth in the dentition an individual "diagnosis".

Gingivitis. This diagnosis is used when gingiva is inflamed, found to bleed on probing. Probing pocket depth and probing attachment level measurements and the radiographic analysis must fail to indicate loss of supporting tissues. "Pseudopockets" may be present.

Periodontitis. Periodontitis occurs when the gum tissues separate from the tooth and sulcus, forming periodontal pockets. Periodontitis is characterized by:

Gum inflammation, with redness and bleeding

Deep pockets (greater than 3 mm in depth) that form between the gum and the tooth

Loose teeth, caused by loss of connective tissue structures and bone

There are different forms of periodontal disease.

Chronic Periodontitis. Chronic periodontitis is the most common type of periodontitis. It can begin in adolescence but the disease usually does not become clinically significant until people reach their mid-30s.

Aggressive Periodontitis. Aggressive periodontitis is a subtype of chronic periodontitis that can occur as early as childhood. It can lead to severe bone loss by the time patients reach their early 20s.

Disease-Related Periodontitis. Periodontitis can also be associated with a number of systemic diseases, including type 1 diabetes, Down syndrome, AIDS, and several rare disorders of white blood cells.

Necrotizing Periodontal Disease. Necrotizing periodontal disease is an uncommon acute infection of the gum tissue. It is characterized by painful and bleeding gums, bad breath, and rapid onset of pain. If left untreated, necrotizing periodontal disease can spread throughout the facial areas (cheeks, jaw) and cause extensive damage. Necrotizing periodontal disease is usually associated with systemic health conditions such as AIDS or malnutrition.

Causes

Periodontal disease is caused by plaque, which is formed from harmful bacteria. The mouth is full of bacteria but they tend to be harmless varieties. Periodontal disease usually develops because of an increase in bacteria quantity in the oral cavity and a change in balance of bacterial types from harmless to disease-causing bacteria. These harmful bacteria increase in mass and thickness until they form a sticky film called plaque.

In healthy mouths, plaque actually provides some barrier against outside bacterial invasion. When it accumulates to excessive levels, however, bacterial plaque sticks to the surfaces of the teeth and adjacent gums and causes infection with subsequent swelling, redness, and warmth.

When plaque is allowed to remain in the periodontal area, it transforms into calculus (commonly known as tartar). This material has a rock-like consistency and grabs onto the tooth surface. Tartar is much more difficult to remove than plaque, which is a soft mass. Once tartar has formed, it must be professionally removed by a dental practitioner.

Periodontal treatment approaches can basically be categorized as:

Nonsurgical Approaches. Scaling and root planning (deep cleaning of tartar and bacteria from gum line and tooth root surfaces), which may include the use of topical or systemic antibiotics.

Surgical Approaches. Periodontal surgical techniques include flap surgery (periodontal pocket reduction), gum grafts, bone grafts, and guided tissue regeneration.

Restorative Procedures. Crown lengthening is an example of a restorative procedure that may be performed for cosmetic reasons or to improve function. For patients who have already lost teeth to advanced periodontitis, dental implants are another option.

Treatment Strategies

In order to determine the appropriate treatment or combination of treatments that best fit their unique situation, patients must work closely with their dental professionals.

Treatments for periodontal disease may include:

Professional cleaning to remove tartar and plaque buildup

Scaling and root planing to the depth of the pockets, sometimes with local anesthesia

Use of a locally or systemically applied antibiotic drug to reduce the bacterial load in the pockets and help lead to reduction of the inflammation

A daily oral hygiene treatment plan for between professional visits

Surgical repair of the diseased tissues

Removal of the damaged tooth

Local antimicrobial delivery into periodontal pockets may be further classified as providing either nonsustained or sustained subgingival drug delivery. Nonsustained subgingival drug delivery provides high pocket concentrations of the antimicrobial agent for only short time periods. Subgingival irrigation with antiseptic agents lacking substantivity for oral tissues (povidone-iodine) is examples of nonsustained subgingival drug delivery. Sustained subgingival drug delivery provides retention of the within periodontal pockets. Controlled drug release can be provided with subgingival irrigation of agents intrinsically substantive for tooth root surfaces (aqueous tetracycline) or pocket placement of commercial antimicrobial fibers, gel or films.

Locally applied antimicrobial agents should be safe, stable, substantive, efficaceous, cost effective, patient compliant, achieve effective concentrations. Factors affecting the bio-availability of an antimicrobial agent are solubility, pH and ion-binding capacity, delivery vehicle-drug interaction and metabolism.

Supragingival irrigation

Home irrigation devices allow the patient to deliver medicaments into the periodontal pocket at home on more frequent basis than is practical with professional gingival irrigation,,the ability of the device to gain a access to the depth of periodontal pocket and the manual dexterity of the patients are the limiting factors . The mechanism of action of irrigation occurs through the direct application of a pulsed or steady stream of water or other solution. Studies have found pulsation and pressure to be critical components of an irrigation device. The pulsation creates two zones of hydrokinetic activity. The impact zone is where solution initially contacts the surface and flushing zone is where solution reaches into the subgingival sulcus. The outcome of hydrokinetic activity is subgingival penetration . Home irrigation devicees include supragingival and subgingival devics. Irrigation with a standard jet tip is generally called supragingival irrigation. Tip is placed coronal to gingival margin. Oral irrigation devices with traditional jet tip results in greater access of medicament to periodontal pocket when compared with rinsing alone. A 90 degree angle of application to the tooth surface provides 71% penetration in shallow pockets. These devices may be useful in delivering of medicaments in cases of gingivitis with shallow pocket depths, they are less useful in delivering medicaments in periodontitis patients with deeper pockets. They are mainly used for full mouth irrigation.

Subgingival irrigation

Irrigation with the soft, site specific tip is often called subgingival irrigation. This also refers to placement of tip, which is placed slightly below the gingival margin. These devices generally include blunt end metal cannula that the patient inserts into the periodontal pocket, this increases the depth of penetration of fluid but has the potential for injury owing to the metal tip. The subgingival tip is generally used for the localized irrigation of specific site, such as a deep pocket, furcation, implant, or crown and bridge. Studies have shown that it can deliver solution into a pocket of 6mm or less up to 90%of its depth. In pockets greater than 6mm, the depth of penetration has been shown to 64%. Professional subgingival irrigation device include a wide range of powered and manually operated irrigations. Irrigation using a syringe with blunt end cannula attached to an oral irrigator can penetrate to 71.5% of the pocket depth in pocket 3.5to 6 mm deep. Vehicles tested for sustained periodontal pocket delivery of antimicrobial agents include solution pastes, hollow fibre, acrylic strips, monolithic fibres, resorbable cellulose, collagen and biodegradable gel

Local antimicrobial agents

A local route of drug delivery can attain 100-fold higher concentrations of an antimicrobial agent in subgingival sites compared with a systemic drug regimen. For example, local placement of a tetracycline- releasing ethylene vinyl acetate monolithic fiber can yield tetracycline concentrations in excess of 1300 Fg/ml in gingival crevicular fluid over 10 days. In comparison, repeated systemic doses of tetracycline- HCl can only provide tetracycline levels of 4-8 pg/ml in gingival crevicular fluid. Disadvantages of local antimicrobial treatment of periodontitis include difficulty in placing therapeutic concentrations of the antimicrobial agent into deeper parts of periodontal pockets and furcation lesions. Personal application of antimicrobial agents by patients as a part of their home self-care procedures is frequently compromised by the patient’s lack of adequate manual dexterity, limited understanding of periodontal anatomy, and poor compliance and performance with recommended procedures.

The task of professionally applying local antimicrobial agents in periodontitis patients with numerous advanced lesions distributed throughout their mouth is time-consuming and labor-intensive. Nonsustained subgingival drug delivery is limited by a only brief exposure of the target microorganisms to the applied antimicrobial agent. Antimicrobial agents locally applied into periodontal pockets do not markedly affect periodontal pathogens residing within adjacent gingival

connective tissues and on extra-pocket oral surfaces (tongue, tonsils and buccal mucosa), which increases the risk of later reinfection and disease recurrence in treated areas. Local agents used for irrigation includes chlorhexidine, povidine iodine, stannous fluoride ,hydrogen peroxide.

Antibiotics

The physical removal of biofilm has proven to be the most effective method for treating periodontal disease. The use of adjunctive antibiotic therapy, either systemic or topical, is controversial. Some studies show superior results with antibiotic use while others show no clinical difference. There is a general consensus that antibiotics should not be used as a monotherapy in the treatment of periodontal disease. Antibiotics as a stand-alone treatment are ineffective at diminishing intact subgingival biofilms.

Antibiotic therapy is generally used as a follow up treatment after conventional mechanical therapy. Aggressive periodontitis may use systemic antibiotics as an adjunctive therapy.

There are many systemic antibiotics on the market. The most commonly used include tetracycline, ciprofloxacin, metronidazole and the penicillins, including amoxicillin and amoxicillin/clavulanate acid (Augmentin®). Tetracycline is bacteriostatic, targets both gram[1]positive and gram negative organisms, and has become bacterial resistant. Ciprofloxacin is bactericidal, targets gramnegative rods, and may cause gastrointestinal discomfort. Amoxicillin and Augmentin are both bactericidal, with Augmentin targeting a more narrow spectrum than amoxicillin. Augmentin was developed due to amoxicillin's bacterial resistance from penicillinase enzyme sensitivity.

Of the many systemic antibiotics available, there is no consensus as to an ideal dose and duration. The choice of antibiotic should be made on an individual basis. In addition to serious adverse effects, like anaphylactic shock, microbial resistance is a growing concern. Other issues with oral antibiotic administration are patient adherence and adequate absorption from the gastrointestinal tract.

Understanding that the periodontal disease process may be initiated by bacteria but the individual's host response was critical to the progression of this disease led to the FDA approval of doxycycline at a sub-antimicrobial dose (20mg twice daily). When administered at this low dose, doxycycline does not cause the long term side effects seen with other systemic antibiotics. Randomized double blind placebo controlled trials demonstrated reduction in probing depths, improvement in clinical attachment levels and decreased bleeding on probing when used as an adjunct with scaling and root planing.

A recent review evaluating non-surgical chemotherapeutic strategies for the management of periodontal disease determined that "systemic antibiotics reach the periodontal tissues by transuduction across serum, then cross the crevicular and junctional epithelia to enter the gingival sulcus." By the time the systemic antibiotic reaches the gingival sulcus it no longer has an adequate concentration to achieve the desired antimicrobial effect. This supports the fact that the mechanical disruption of biofilm must be included in the treatment of periodontal disease.

Atridox® is a 10% doxycyline hyclate gel and is prepared by mixing powder and liquid from two syringes. The antibiotic is administered into the gingival sulcus through a cannula. Absorption lasts up to 21 days, while therapeutic drug levels in the gingival crevicular fluid start to decline at 7 days. The most notable drawback is the high level of clinician skill needed to deliver this therapy as the material tends to come out of the pocket as the syringe is being pulled out of the sulcus. The majority of the time, more than one site can be treated depending on the depth and size of the pockets.

Arestin® is comprised of spheres embedded with 2% minocycline HCl that is slowly released and holds the therapeutic dose in the gingival crivicular fluid for 14-21 days. The most notable drawback for Arestin is the delivery dose. The syringe holds pre-set doses that may not be sufficient for every site. This results in the need to reapply in the same pocket.

Currently, resorbable antibiotics such as Atridox® and Arestin® are the topical antibiotics of choice.The American Academy of Periodontology (AAP) supports that local adjuncts, when compared with scaling and root planning alone, provide limited improvement. Locally administered antibiotics still require a strict health history review to verify there are no known allergies. Even though these medications are applied topically, as opposed to oral administration, the same precautions apply.

Antiseptics

Unlike topical controlled-released antibiotics, oral rinses do not penetrate deep into the gingival sulcus. Despite this limitation they do show benefit when used adjunctively for gingival inflammation. Oral rinses are also of great value in post surgical healing. Substantivity is a crucial component when considering the effectiveness of a mouthrinse. This term refers to the adherent qualities of a mouthwash and its ability to be retained. Saliva has a natural flushing property making it difficult to maintain an antimicrobial effect. Research shows a significant antibacterial effect up to 7 hours after mouthrinses with high a substantivity property.

First generation antimicrobials include phenolic, sanguinarine, qua- ternary compounds. Listerine® and its generics are phenolics, which possess the only ADA Seal of Acceptance among the first generation antimicrobials. Listerine contains 26.9% alcohol, alters the bacterial cell wall, and has 36% gingivitis reduction. Cepacol® and Scope®, quaternary ammonium compounds, contain 14% and 18.9% alcohol respectively, increase bacterial cell wall permeability causing cell lysis, and reduces gingivitis approximately 15%.1

Second generation antimicrobials include cetylpyridinium chloride (CPC) and chlorhexidine (CHX). A commercial name for CPC is Crest® Pro-Health®, which contains 0.07% CPC. Bacteria cells are killed by cellular pressure, resulting in a similar efficacy as Listerine. Chlorhexi- dine has many commercial products including the availability of a nonalcoholic version by Sunstar Americas, Inc. Peridex® by 3M Espe and Periogard® by Colgate® Professional are two examples of popular chlorhexidine-based products. Their active ingredient is 0.12% chlorhexidine. Cell death results from altered osmotic equilibrium. CHX efficacy in the reduction of certain aerobic and anaerobic bacteria has been shown to be as high as 97% after 6 months of use. CHX has 29% gingivitis reduction. The gingivitis reduction percents listed above for both first and second generation antimicrobials are based on efficacy data published by manufacturers.

Other antimicrobials include oxygenating, chlorine dioxide, and zinc chloride agents. Peroxyl® is an oxygenating agent with the active ingredient of hydrogen peroxide. It has anti-inflammatory properties as well as a bubbling action to clean and alleviate discomfort. Short term studies have produced controversial findings. Oxyfresh®, a 1% chlorine dioxide agent, has minimal plaque reduction. It is a stable, free radical and an oxidant with algicidal, bactericidal, cysticidal, fungicidal, sporicidal, and viricidal properties. Oxyfresh is primarily used for the treatment of halitosis. Breath Rx® is a zinc chloride agent designed to odorize sulfhydryl groups with zinc ions. It claims to be a scientific bad breath treatment specifically designed to help treat the causes of bad breath and the symptoms.

Antimicrobial mouth rinses have been linked to several side effects; some more serious than others. First generation compounds like Listerine can cause a burning sensation and bitter taste.

Chlorhexidine can cause supragingival calculus build-up and staining. Research has demonstrated permanent damage to enamel through erosive pH levels and abrasive antimicrobial toothpastes.1 Carcinogenic changes have been linked to the use of oxygenating agents and mouth rinses containing alcohol.

Nutraceuticals

As antibiotic resistance becomes more of a concern, health care pro- viders looking for alternate adjunctive periodontal therapies for their patients. Some examples of nutraceuticals include herbal and nutri- tional supplements and the future of this type of therapy is promising. There are approximately 500,000 plant species, with only 1% having been photochemically investigated. Herbal plant extracts have been shown to reduce the level of biofilms influencing the level of bacterial adhesion. This has shown results with the reduction of periodontal disease. Some herbs such as Coptidis rhizome extract and Hamamelis virginiana, are used as bactericidal agents against oral bacteria while others such as cranberry, Polygonum cuspidatum and Mikania are used to inhibit adhesion.

The use of probiotics in the control of periodontal pathogens is emerging. Probiotics are "live microorganisms, which when administered in adequate amounts confer a health benefit on the host." Simply put, they are healthy bacteria that displace unhealthy or pathogenic bacteria. A reduction in gingivitis and dental plaque has been shown with the administration of L. reuteri Prodentis® gum chewed twice daily in patients with moderate to severe gingivitis.14 GUM® Perio Balance®, marketed by Sunstar Americas, is a once daily lozenge with L. reuteri Prodentis® that claims a reduction in moderate to severe plaque and bad breath. EvoraPlusTM from Oragenics, Inc. is another new probiotic for oral health and is used once daily. This supplement contains a combination of three bacterial strains Streptococcus uberis KJ2, S. oralis KJ3, and S. rattus JH145, and claims a reduction in periopathogens within the periodontal pocket.

Physiotherapy is performed after the removal of dental deposits, arresting of acute inflammatory process. Some procedures (UV-radiation, hydrotherapy, laser therapy, aeroionotherapy, etc.) can sometimes be performed from the very beginning of the complex treatment.

Various physiotherapeutic methods are used for treatment:

Electrotherapy: a) direct current (electrophoresis, vacuum electrophoresis) b) pulse current of low frequency and low voltage (diadynamotherapy, fluctuorization); c) pulse currents of high (HF), ultrahigh (UHF) and super-high (SHF) frequency (darsonvalization, diathermy, diathermocoagulation, electrorhythm therapy, UHF-therapy, microwave therapy).

Ultrasound therapy.

Aeroionotherapy

Phototherapy (UV light therapy, laser therapy).

Vacuum therapy.

Hydrotherapy.

Massage.

Heat and cold treatment.

Magnetic therapy.

Oxygen therapy.

The above list does not cover all currently available physiotherapy procedures, which are constantly invented.

Direct current.

Electrophoresis, a method of administration of drugs into body tissues using a direct electric current, is often used in the treatment of periodontal diseases. Depending on the place of drug administration, dental, supragingival and intranasal electrophoresis; ionic (galvanic) "collar" according to Scherbak, etc. is distinguished. In electrophoresis, the active electrodes are applied to the gingival margin through a hydrophilic pad soaked in the drug substance. The passive electrode is fixed in the hand or on the forearm. The pad of the passive electrode is moistened with tap water or isotonic sodium chloride solution. Electrophoresis is prescribed in acute and exacerbated chronic catarrhal and hypertrophic gingivitis, periodontitis after elimination of active inflammation, and parodontosis.

In hypertrophic gingivitis electrophoresis with 10% solution of calcium chloride alternately from an anode and a cathode is recommended. This provides the deposition of calcium and chlorine ions in the gum tissue for anti-inflammatory (calcium) and cytocidal (chlorine) action, which ensures a lasting clinical effect. electrophoresis with ascorbic acid (5%), vitamin P (1%), solutions of trypsin, ribonuclease (1 mg / mol) from the anode and aqueous solution of aloe extract, 1% solution of nicotinic acid, vipraxin, heparin solution (1:15) from the cathode, mud extract, ozokerite, etc. is used to influence the exudative processes.

In parodontosis, electrophoresis with 1-2-4% of solution of sodium fluoride, 2,5% of solution of calcium glycerophosphate is recommend and in hyperesthesia of hard tooth tissues electrophoresis with solutions of thiamine chloride with novocaine is advisable. Electrophoresis with drugs, listed above, improves mineral metabolism and trophism of periodontal tissues. If it is necessary to administer complex drugs consisting of differently charged ions, electrophoresis is carried out alternately on one day from the negative, in another - from the positive pole.

Diadynamotherapy is the use of modulated sinusoidal pulse current for therapeutic purposes. The mechanism of physiological action of diadynamic current is based on the redistribution of ions in tissues, changes in the permeability of membranes and cell membranes, improvement of blood circulation, trophism, analgesia, etc. This increases the protective properties of tissues; they accumulate biologically active substances (heparins, histamine-associated substances, etc.), and others. In the treatment of periodontal disease, the most appropriate use of diadynamic current is with the simultaneous administration of drugs into the tissue (diadynamophoresis).

Fluctuorization is the use of alternating current with randomly varying forces, frequency and duration of oscillations for therapeutic purposes. For these purposes, three forms of current are used: bipolar symmetric, partially rectified and direct pulsating current. The first two forms of current have anti-inflammatory and analgesic effects. Constant pulsating current can be used for electrophoresis with drugs (fluctuorophoresis). Fluctuorization is indicated in the treatment of acute forms of gingivitis, periodontitis and parodontosis.

Alternating current.

Darsonvalization is electrotherapy, performed using devicesgenerators of high-frequency alternating current of low power, high voltage, which gives a sharply attenuating discharge. D'Arsonval currents suppress the sensitivity of peripheral nerve receptors, giving the analgesic effect, reduce itching in tissues, dilate blood vessels, improve tissue trophism, relieve vascular

spasm, increase WBC migration, etc. Darsonvalization is indicated in chronic gingivitis, generalized periodontitis, parodontosis.

Electrorhythm therapy is based on the use of a low-power output sinusoidal current with a frequency of 2000 Hz and modulated at a low frequency (5-100 Hz) in the form of sawtooth rhythmic pulses. Currents are used in two modes: two-stroke and rectified. The method of electrorhythm therapy has a stimulating, electrophoretic and pronounced analgesic effect on tissues; provides a deeper diffuse and prolonged effect of drugs, especially on the hard tooth tissues and skin.

UHF-therapy is one of the types of electrotherapy. The mechanism of physiological action of the ultrasonic field is provided by the physic-chemical changes in the colloids of molecules closely related to thermal and oscillatory effects. There are athermic, oligothermal and thermal doses. The field of weak intensity stimulates, and strong - suppresses the functional activity of the organism. Under the influence of the UHF field, the capillaries dilate, their blood circulation accelerates, macrophage activity increases, tissue acidity decreases, edema decreases, metabolic processes improve, the growth of young connective tissue accelerates, the sensitivity of nerve receptors decreases, and so on. UHF therapy is used in acute gingivitis, exacerbation of periodontitis, abscess.

Microwave therapy is the use of electromagnetic field of the microwave range. Microwaves are able to penetrate into the body tissue to a depth of several centimeters. At the sites of penetration the energy is absorbed by the structures according to their density and converted into endogenous heat. Under the influence of microwaves, blood vessels dilate, blood circulation accelerates, trophism and metabolism become to normal, the sensitivity of nerve endings is reduced, the regeneration process is stimulated, and so on. The non-thermal effect of microwave therapy is manifested in various intramolecular physicochemical reactions. At a dose of 5-7 watts at an exposure of 5-7 minutes, microwaves have antiinflammatory, antispasmodic and analgesic effects. Indications for microwave therapy are the same as for UHF therapy.

In the treatment of periodontal disease ultrasound, which enhances metabolism, activates the activity of enzymes, increases the permeability of the membrane, while releasing biologically active substances, is also used. The effect of ultrasound on tissues is considered as a kind of micromassage of cells. Ultrasound is used to administer drugs into tissues, phonophoresis (e.g., vitamins D, E, group B, sodium fluoride, etc.). Ultrasound oscillations from 0.8 to 20 MHz can be used to diagnose tissue damage (ultrasonic bioecholocation). This is a lifelong method of studying the structure of periodontal bone tissue. It allows evaluating the density of tissues. The method is successfully used to diagnose and estimate the quality of treatment. Ultrasound is also used to remove dental deposits. Through the contact acoustic environment, namely, water, dental deposits are effectively and easily removed by the light massaging movements of the tip in the surface of the tooth neck and crown.

Aeroionotherapy. Aerosol method of drug administration is used in acute inflammatory diseases and exacerbation of chronic periodontal diseases. The principle of operation of inhalators is based on spraying drugs with a stream of compressed air into small particles that penetrate freely into the periodontal tissue. Due to the huge absorption capacity of the mucous membrane of the mouth and respiratory tract, aerosol inhalations, in addition to local action on periodontal tissue, have a general resorptive effect: improve lymph and blood circulation, activate metabolism.

Phototherapy. Ultraviolet radiation has anti-inflammatory, desensitizing, stimulating metabolic processes and regenerating effect; it is widely used in prominent inflammation of periodontal tissues. The most effective are short ultraviolet rays (253.7 nm). Conical metal nozzles

make it possible to direct the rays locally into the oral cavity. Laser therapy is the use of electromagnetic waves emitted by quantum generators, with monochromaticity, coherence (single-phase waves). Helium-neon laser radiation is the most widely used. Such radiation has a therapeutic effect of a wide range: antiinflammatory, because it normalizes the disturbed microcirculation and permeability of the vascular wall; analgesic; thrombolytic; improves metabolic and redox processes in tissues; stimulates the regeneration process, factors of local and general immune protection of the body, etc. Intravenous laser irradiation of blood is used in combination with other prescriptions in the treatment of severe forms of generalized periodontitis, ulcerative necrotic stomatitis. The method reduces the duration of treatment, increases the remission period, has an analgesic effect, stimulates regeneration processes.

Contraindications are diseases with severe clinical course, cardiovascular diseases (myocardial infarction, aortic aneurysm, circulatory failure of II-III degree), tuberculosis intoxication, diabetes mellitus at the uncompensated stage, blood diseases.

Vacuum therapy. The essence of treatment is the formation of hematomas on the gums in the area of the transitional fold as a result of exposure to reduced pressure (up to 40 mm Hg). At each session, 4-6 hematomas are formed on different parts of the gums, which, when resorbed, act as biogenic stimulants, activating trophic, immunobiological and regenerative processes. In periodontal tissues, conditions are created for the arrest of the inflammatory process, etc. Indications are chronic catarrhal and hypertrophic gingivitis, occurring with congestion, generalized periodontitis without discharges from the periodontal pockets and parodontosis.

Vacuum massage is a type of vacuum therapy. In areas of short-term vacuum in the gums and the transitional folds of the mucous membrane of the vestibule of the mouth, microhematomas are formed. The process of moving of the vacuum areas throughout the periodontium is a kind of massage. As a result, blood circulation in periodontal tissues improves, stagnation and hypoxia are eliminated, metabolism, redox processes are improved, and regeneration processes are activated.

Hydrotherapy. Hydrotherapy is the irrigation of the oral cavity with various saturated liquids or aqueous solutions from special devices and apparatus at a pressure of 1.5-2.0 atm. Under the influence of hydrotherapy, the receptor apparatus and capillary network are simultaneously irritated and the aerosol effect of the drugs used is manifested. Tinctures and decoction of medicinal herbs (chamomile, sage, kalanchoe, eucalyptus, plantain, etc.) are the most effective, as well as sea water, and mineral waters; water saturated with carbon dioxide, oxygen; water subjected to magnetic processing; light solutions of furacillin, hydrogen peroxide, dimexid, galascorbic acid, 2% citral solution, etc. For irrigation special nozzles are used: hollow tubes, curved along the dental arch with many small holes. The jets of fluid coming through the holes of the nozzle on the gums, create different levels of impact on the tissues, improve blood circulation, trophism, therapeutic and prophylactic effect.

Massage. Vibration massage is a rhythmic repetition of vibration movements on the surface of the areas. Such type of massage improves blood circulation, metabolic processes, tissue trophism, accelerates regeneration processes. Special vibrating massagers and vibrating toothbrushes of industrial production are use. There are many attachments to the dental tip that vibrate during the operation of the drill. The massage facilitates active hyperemia of the gums, the capillary network dilates and the blood circulation accelerates, the metabolic processes in the periodontal tissues improve, the phenomena of hypoxia are eliminated. Massage improves lymph flow, which promotes the resorption of inflammatory exudate, reduces tissue swelling. Under the influence of massage, the protective properties of periodontal tissues are enhanced.

Digital automassage improves blood and lymph circulation, stimulates metabolism and increases gas exchange in periodontal tissues. This type of massage leads mechanical irritation of numerous receptors of gums which include the reflex mechanisms causing therapeutic effect. The gums are massaged with the forefinger, placing it on the transition fold at the base of the interdental papilla. Then make a movement of the finger to its top - from 6 to 10 movements, in each area covering 2-3 papillae of the gums. Finish the massage with a hygienic rinse. Contraindications are exacerbated course, the presence of abscesses, erosions, canker sores, ulcers, neoplasms.

Heat treatment. Deep heating of heat treatment takes an important place in the complex treatment of periodontal diseases. Curative mud has an effect on periodontal tissues due to its chemical, physical and biological properties. Sulphide mud, peat, sapropels are used for medical purposes. The therapeutic effect of mud applications is based on a complex reflex process. Biogenic stimulants and microelements that affect sensitive receptors are absorbed through the mucous membrane and vascular and metabolic processes are stimulated. Active hyperemia develops, outflow accelerates, the pH of the medium changes to the alkaline side. Paraffin and ozokerite treatment is one of the types of heat treatment. Paraffin is a mixture of high molecular weight carbohydrates with high heat capacity and low thermal conductivity. It has a pronounced thermal and compression effect. Ozokerite, or the earth wax, whose melting point is 52-68 ° C, has compression and thermal properties. To increase the ductility of paraffin and ozokerite in the heating process, you can add Vaseline or Vaseline oil. It is applied to the area of the pathological focus. It has a soothing, anti-inflammatory, analgesic and antispastic effect.

Cryotherapy. Cryotherapy, or local hypothermia, is a method of treatment based on the use of low temperatures. Under their influence on the focus of lesion complex physical, chemical and biological processes take place in periodontal tissues: painful sensitivity decreases, tissue swelling decreases, protein decomposition and processes of absorption of decay products of tissues and microorganisms are slowed down, hypoxia and acidosis, formation, secretion and secretion decrease, the phagocytic activity of leukocytes increases, reparative processes are stimulated; a rupture of the cell membrane occurs due to intracellular crystallization of water, denaturation of cellular proteins until cell death, and others. Advantages of cryotherapy are painlessness, limited destructive effect, pronounced hemostatic effect, favorable clinical course of the wound process with the formation of a delicate scar. Liquid nitrogen, freon, etc. are used as freezing liquids. The complex treatment of periodontal diseases includes methods of cryotherapy, contact cryodestruction and cryo-curettage.

Magnetic therapy. The constant magnetic field has an anti-inflammatory, antispasmodic, analgesic effect on the human body, accelerates reparative processes and others. For the treatment of periodontal diseases, dentogingival caps and toothbrushes with built-in permanent magnets are used. Permanent magnets are sources of a constant magnetic field, the magnetic field lines of which are concentrated in the pathology zone. The magnetic cap is periodically used by patients for 20-30 days. Under the influence of the magnetic field, swelling, redness, bleeding gums, exudate from periodontal pockets decrease. The magnetic toothbrush provides daily magnetic massage of periodontal tissues. A combination of electrophoresis, laser therapy in combination with magnetic therapy is effective. Applications, irrigation, rinsing, mouthwashes, hydromassage with pre-magnetized drugs can be recommend.

Oxygen therapy. Periodontal diseases are accompanied by varying degrees of oxygen starvation. This condition also contributes to the depletion of vitamin C, increase vascular permeability, disorders of all types of metabolism. As a result of hypoxia deoxidized metabolic products accumulate in the periodontal tissues, resulted in chronic tissue hypoxia. Disorders of energy metabolism are one of the pathogenetic links in the development of dystrophic changes in

periodontal tissues. Consequently, the local administration of oxygen into periodontal tissues is one of the pathogenetic methods of treatment and is widely used in clinical practice. The simplest methods are tissue saturation with cotton swabs soaked in hydrogen peroxide, potassium permanganate, irrigation or aerosol spraying with these solutions that is a hydrotherapy when the water jet or drug solution is enriched with oxygen (oxygen baths, oxygen punches, etc.).

The use of physiotherapy methods (namely electrotherapy) is contraindicated in malignant neoplasms, benign tumors of the maxillofacial area, decompensated forms of diseases of the cardiovascular, respiratory and endocrine systems; blood diseases, active tuberculosis, pregnancy.

## TOPIC 58.

## DIFFERENTIAL DIAGNOSTICS OF TRAUMATIC INJURIES OF ORAL MUCOSE. MODERN METHODS OF TREATMENT AND PREVENTION OF TRAUMATIC INJURIES OF ORAL MUCOSE.

Chronic mechanical injury (trauma mechanicum chronicum)

Complaints:

sensation of discomfort, pain, swelling; frequent biting;

• presence of the old ulcer; growth in the hard palate, the gums, the tongue, under the prosthesis; whitish section of the mucous membrane in the place of the permanent trau matic factor.

• Lymph nodes are increased, painful during palpation. The manifestation of changes depends on gravity of the lesion. Inflammatory spot or erythema in the place of the injury. • Localization of erosion corresponds to the traumatic agent, erosion is painful, localized on the hyperemized mucous membrane. • Indolent ulcer is localized more frequently on the tongue, lips, cheeks along the line of teeth joining, and also in the limits of orthopaedic field. It is sin- gle, painful, surrounded with inflammatory infiltration, the bottom is most often uneven, covered with fibrinous fur. The decrease, the disappearance of painfulness, the appearance of papillary growths indicate about malignization.

Clinic:

Papillomatous hyperplasia – papilloms with the soft, grainy, bright red sur- face under the prosthesis, they are more frequent localized in the region of the hard palate. • With the habit to bite or to suck lips, tongue, and cheeks mucosa membrane (in essence along the line of teeth joining) acquires the unique form: it will swell, it has the white macerated surface in the form of either spots and large illegibly limited sections, or fringed form because of many small patches of the unevenly biting epithelium. Lesion has asymptomatic course, but during the deep removal the erosions are formed, they are painful in contact with chemical stimuli.

Treatment:

1. The elimination of the traumatic agent.

2. Anesthetization.

3. Cleaning the surface of erosion and ulcer from the necrotic fur. 4. Processing of ulcer and oral cavity by the solutions of the antiseptics. 5. Stimulation of the epithelisation.

Physical and mechanical traumas of oral mucosa

Linea alba (white line)

Localization: Buccal mucosa, at the level of the occlusal line of the teeth. It is a horizontal streak on the buccal mucosa at the level of the occlusal plane extending from the commissure to the posterior teeth.

Clinical features: Lesions are mostly asymptomatic. The common visual symptom of linea alba is the presence of whitish, linear, filament-like plicae formations, horizontally parallel to the occlusal level of bicuspids and molar teeth in both left and right sides of buccal mucosa. Palpation should give a tactile sensation of normal mucosa texture. It is more prominent in individuals with

reduced overjet of the posterior teeth. It is often scalloped and restricted to dentulous areas. The diagnosis is based on clinical grounds alone.

Etiology: Lesions mainly arise from occlusal traumas of posterior teeth generated due to the parafunctional cheek sucking of patient. The sucking habit is also associated with friction between buccal tubercules and irritates the buccal mucosa by pressure. Prevalence of such lesions is about 6.2–13% in the population.

Treatment: No treatment is required; the white streak may disappear spontaneously in some people. But very sharp-edged teeth can be corrected.

Chronic biting (Morsicatio buccarum)

Localization: The lesions made by chronic bite trauma (nibbling) on the buccal mucosa generally cause keratinized shreds, tissue tags, or erosive and desquamative surfaces. These lesions according to their localizations are called as “morsicatio buccarum” if they are localized on the buccal mucosa, “morsicatio labiorum” if they appear on the labial mucosa, and “morsicatio linguarum” if they occur on the lateral borders of the tongue. The lesions are seen on the buccal mucosa, bilateral chewing line, labial mucosa, and lateral edges of the tongue.

Clinical features: Lesions are apparent as shallow whitish wrinkles which are diffuse and present irregularly on the buccal, labial mucosa, and tongue. Epithelial desquamation occurs on the surface. In some cases, erosions and petechiae may be seen. The lesions could be diagnosed by clinical inspection.

Etiology: It is often related to chronic biting of the oral mucosa seen in psychologically tense patients. Parafunctional bite of the buccal mucosa, lips, and tongue until wear of superficial epithelium and wound formation is consciously made by those patients. The incidence of morsicatio buccarum was reported to be 2.5% in Caucasian populations.

Treatment: Treatment is usually unnecessary. It is recommended to stop the habit. Psychological treatment can be suggested for stopping a bad habit. Acrylic splint can be made on the occlusal surface of the teeth. It is accepted as a precancerous lesion.

Traumatic ulcers

Localization: Presence of traumatic ulcers is a relatively common finding in dental practice. Such lesions arise from trauma related to bite of buccal mucosa, lateral border of the tongue or lips during chewing. Traumatic ulcers seen in the mucobuccal folds and gingiva are related to different irritant factors such as hard foods and inappropriate hard brushing. Traumatic ulcer due to lip biting after inferior dental nerve block is seen on the lower lip. During orthodontic treatment, traumatic ulcers can occur especially on the buccal mucosa due to the irritation of braces or appliance wires.

Clinical features: Traumatic ulcers could be of solitary shallow or deep discontinuity type showing on the epithelium and are associated with peripheral keratosis of mild to severe degree. The bottom of the ulcerative lesions is made of whitish or yellow pseudomembrane. Upon elimination of the causative factor, often the ulcer heals with or without scar depending on the extent of the damage occurred.

Etiology: They could originate from accidental mucosal biting, sharp edges of prosthesis, sharp or pointed food stuff, during orthodontic treatment, lip biting after injection of local anesthetic solutions, neonatal teeth, or faulty tooth brushing. During dental treatments, iatrogenic damages can result in traumatic ulcer formation. Some medical treatments can cause oral ulcerations, such as brutal intubation for general anesthesia, ENT surgeries, or endoscopic interventions and iatrogenic

malpractice applications. A high prevalence of traumatic ulcer of about 21.5% was reported among lower classes of Brazilian population. Most prevalent types of lesions were reported to be traumatic ulcer and actinic cheilitis (7.5% for each). Among the etiological factors of traumatic ulcers could be mentioned traumas caused by bites, dental appliances, inappropriate tooth brushing, misfit of removable partial or total dentures, irritating caries edges, malocclusion and puncturing restorations.

Treatment: Most often, traumatic ulcers can heal spontaneously and uneventfully without complications in a brief period of time. But, in case of persistent traumatic factors, such as presence of sharp tooth morphology, cutting edges of restorations, and puncturing appliance contours, especially inadequate surfaces of removable prosthesis, continuous trauma arising from above-mentioned causes can lead to formation of chronic ulcers.

Chemical injuries of the oral mucosa

Chemical burn

Localization: Gingiva and mucobuccal folds are main localization regions of such lesions.

Clinical features: The wounds have irregular shape and white color, are overlaid by a pseudomembrane, and are very painful. Lesions can cover an extended area. If the lesions are contacted shortly, a shallow whitish and wrinkled appearance occurs. Brief contacts cannot cause necrosis.

Etiology: Caustic chemical and drug materials when they come in contact with the oral mucosa are often very irritating and cause direct mucosal trauma. Inappropriate usage of medications, such as aspirin application onto the neighboring mucosa of painful teeth with decay, may result in mucosal trauma. Iatrogenically, during dental treatments irrigant solutions (sodium hypochlorite or formalin) or some endodontic pastes with arsenic can irritate the mucosa. However, such injuries are not very common since the introduction of rubber dam in dental practice.

Treatment: The best treatment of chemical burns of the oral cavity is prevention. The proper use of a rubber dam during endodontic procedures reduces the risk of iatrogenic chemical burns. Superficial burns of mucosa can heal in a short period of time (within 1 or 2 weeks) as the turn-over of oral mucosa is very high. Oral surgery and antibiotics are necessary in very rare cases. Gel with hyaluronic acid can accelerate the healing process. Possible treatments after chemical injuries, in relation with the severity of wounds, are topical and intralesional corticosteroid applications, caustic acid ingestion, commissuroplasty, mucosal flaps, free radial forearm flap and free jejunal graft, surgeries made with electrocautery or soft tissue laser, and wound coverage by periodontal pack

Contact allergic stomatitis

Localization: Contact area of oral mucosa due to denture base materials, restorative materials, mouthwashes, dentifrices, chewing gums, food, and other substances. Various chemical or natural agents in contact with the mucosa can irritate and cause contact stomatitis. For example, cinnamaldehyde or cinnamon essential oil, which are commonly used as flavoring agents in foods, beverages, candies, and hygiene products by contact with mucosal surfaces, may trigger the formation of allergic stomatitis.

Clinical features: Diffuse erythema, edema, occasionally small vesicles, and shallow erosions appear immediately after contact with the allergen on the affected mucosal surfaces. Lesions are associated with burning symptom. In chronic allergies, whitish, hyperkeratotic, erythematous lesions form .

Etiology: Denture base materials, restorative materials like amalgam, mouthwashes, dentifrices, chewing gums, food, and other substances may be responsible.

Treatment: Contact allergic stomatitis can be diagnosed by an accurate examination and clear understanding of medical history of the patient. Clinician's diagnostic ability and experience are highly important to avoid further unnecessary examinations, invasive and expensive diagnostic procedures. Treatments include removal of suspected allergens, and use of topical or systemic corticosteroids, antihistamines.

Radiation injuries

Oral mucositis

Localization: Developments in oncology have led to improved survival rates for different cancers. Unfortunately, those treatment regimens have side effects such as formation of oral mucosal lesions. The most common wound type during chemotherapy is oral mucositis which appears by inflamed erosive or ulcerative lesions on mucosal surfaces in the oral cavity. Generally, buccal mucosa is affected by radiation treatment of head and neck tumors.

Clinical features: After radiotheraphy, at the end of first week, the first oral manifestations can appear. Lesions are erythematous and edematous. In the following days, ulcerative erosions with whitish-yellow exuadate appear. As salivary glands are involved, xerostomia occurs and is followed by tongue papillary changes with loss of taste, burning sensation, and pain during function. Speech is also affected negatively.

Etiology: Chemotherapy, radiotherapy, or their combinations can lead oral mucositis. The majority of patients (approximately 20–40%) receiving conventional chemotherapy regimens for solid tumors, in relation to the dose and cytotoxicity of the drug used, have oral mucositis. It is a side effect of radiation treatment of head and neck tumors.

Treatment: Supportive care, cessation of radiation treatment, B-complex vitamins, and sometimes low doses of corticosteroids are suggested.

Electrical and thermal burn

Electrical burn

Localization: Commissures of the mouth often result in severe facial scarring and deformation. Most commissural electrical burns involve mucosa, submucosa, muscle, nerve, and vascular tissue.

Clinical features: Damage made accidentally to lingual or/and labial arteries can cause abundant bleeding. When burned tissues spontaneously start to loosen or slough and occasional trauma occurs, this type of bleeding happens. Generally, this is observed 3–4 days after burn injury. Pressure should be applied to the hemorrhage site to stop the bleeding and the patient should be taken to the nearest hospital emergency room for definitive care.

Etiology: The majority of electrical burns are home accidents. Generally, children play with live electric extension cables/cords and may contact or suck them and are injured by current. Especially in the cable/plug junctions, in non-fitted appliance plugs, the electric current flows through tongue or oral cavity when they are in contact with saliva, and the electric energy burns oral tissues. Children under three years of age are mostly affected by this type of injury.

Treatment: Whatever is the severity of burned injury, the basic treatment strategy involves pain relief, infection control, and acceleration of wound repair. Application of antibiotic ointments

to the burn area has been recommended by some authors. Systemic antibiotics are recommended by most clinicians to prevent wound infection. Facial disfigurement takes place if splints are not applied. Microstomia reduces mouth opening, renders oral hygiene difficult, and decreases functions of speech and chewing. Most of the cases need plastic surgery.

Thermal burn

Localization: Oral mucosa, especially tongue and palatal mucosa.

Clinical features: Clinically, the condition appears as a red or white, painful erythema that may undergo desquamation, leaving erosions. In excessive damage to tissues, necrosis could appear. In mild lesions, wounds can heal spontaneously within a week.

Etiology: Thermal burns mostly happen by accidental ingestion of hot substances. High incidence of thermal burns with a prevalence of 24.6% is seen among children and young patients. Usually caused due to contact with very hot foods, liquids, hot metal objects or iatrogenic usage of lasers (diodes, Nd:YAG, Er:YAG or CO2), piezoelectric surgery, or electrosurgery devices.

Treatment: No treatment is required for simple lesions. Care should be taken in deep lesions to avoid contamination during healing period. Saline would be prescribed to accelerate wound healing and avoid bacterial ingrowth. Ozone therapy and laser biomodulation could help for good prognosis. In severe damages, prophylactic antibiotic coverage is recommended. In hard tissue damages related to thermal burn, the necrotic area should be removed surgically in order to avoid damage to surrounding vital tissues and obtain blood supply for repair and subsequent regeneration.

## TOPIC 59.

## DIFFERENTIAL DIAGNOSIS OF PRIMARY (AUTOINFECTIVE) LESIONS OF ORAL MUCOSE. ACUTE HERPETIC STOMATITIS. MODERN METHODS OF TREATMENT AND PREVENTION

Stomatitis is inflammation of the mouth. It affects the mucous membranes

Stomatitis is a type of mucositis, a condition defined as pain or inflammation of the mucous membrane.

Mucositis is a relatively common side effect of chemotherapy and sometimes radiotherapy. It can affect the inside of the lips, cheeks, gums, tongue, and throat.

Types

There are two main types of stomatitis:

Canker sores

These are also known as aphthous ulcers and are part of the most common cause of stomatitis. The sores are pale white or yellowish in color with a red outer ring.Canker sores can develop singly or in a cluster and usually occur on the inside of the lips or cheek, or on the tongue.

Cold sores Cold sores are small, painful, fluid-filled sores that usually occur on or around the lips near the edge of the mouth. Caused by the herpes virus (HSV), the condition is also known as herpes stomatitis.Stomatitis can be broken down into different categories, depending on which area of the mouth is affected:

cheilitis – inflammation of the lips and around the mouth

glossitis – inflammation of the tongue

gingivitis – inflammation of the gums

pharyngitis – inflammation of the back of the mouth

The most common causes are:

trauma from ill-fitting dentures or braces, biting the inside of the cheek, tongue, or lip, and surgery

Other examples include: bacterial infections sexually transmitted infections weakened or deficient immune system irritation from strong chemicals stress certain diseases, including Behcet's disease, Crohn's disease, and lupus medications, including sulfa drugs, anti-epileptics, and some antibiotics nutritional deficiencies allergic reactions burns caused by hot food and drink

It is important to identify the cause of stomatitis in order to treat it properly.

Symptoms

Stomatitis often results in pain, stinging, and soreness. Each person may experience different symptoms. These can include mouth ulcers with a white or yellow layer and red base, usually inside the lips, cheek, or on the tongue

red patches

blisters

swelling

oral dysaesthesia

a burning feeling in the mouth

lesions that heal in 4-14 days and often recur

Diagnosis

Diagnosis will depend entirely on what is causing the stomatitis. Relevant investigations include a physical examination, as doctors can learn a lot by looking at the appearance and distribution of ulcers.

## TOPIC 60.

## DIFFERENTIAL DIAGNOSTICS OF PRIMARY (AUTOINFECTIOUS) LESIONS OF ORAL MUCOSE. ACUTE ULCERATIVE NECROTIC STOMATITIS OF PRIMARY STOMATITIS. MODERN METHODS OF TREATMENT AND PREVENTION.

Auto-infectious diseases result from the action of conditionally pathogenic microorganisms that vegetate in the oral cavity in the reduced reactivity of the mucous membrane and the body as a whole. Contributing factors for the emergence of autoinfectious stomatitis (acute aphthous and herpetic, acute ulcerative necrotic stomatitis) are insufficient oral hygiene, malnutrition, trauma to oral mucosa, retention of the wisdom tooth, hypothermia. These diseases are most often found at the outpatient reception of the dentist, they have a moderate and severe course, require comprehensive examination and carefully conducted differential diagnosis. Therefore, deepening clinical thinking of doctorssubordinates is essential for appropriate prescription of medications for comprehensive treatment and preventive measures.

Primary stomatitis Inflammatory diseases of oral mucosa, the etiological factor of which acts directly on the mucous membrane of the oral cavity

Autoinfectious stomatitis Arising from the action of conditionally pathogenic microorganisms that vegetate in the oral cavity in the reduced the reactivity of the mucous membrane and the body as a whole

Acute catarrhal stomatitis Exudative inflammation of the mucous membrane, characterized by hyperemia, increased desquamation of epithelial cells, increased discharge of leukocytes and serous exudate from the mucosa, and an increased amount of mucus in the mouth

Stomatitis ulceronecrotica Vincenti. It has a course by the type of hypergeneric inflammation, expressed signs of alteration of oral mucosa. This condition is characterized by the formation of ulcers, bleeding gums, severe pain in the lesions, fever and intoxication of the body. The disease occurs at the age of 18-30, more often in men. The development of the disease is facilitated by hypothermia, traumas of oral mucosa, violation of hygiene rules. In the pathogenesis of great importance is the sensitization of the body to streptococcal microflora. The course of stomatitis has signs of an infectious disease. Patients complain of headache, lethargy, subfebrile body temperature, joint aches. In the mouth, there are bleeding gums, burning sensation and dryness of oral mucosa. In the next phase of detailed clinical manifestations, patients complain of an increase in general weakness, fever, headache, and reduced performance. Pain in the oral cavity is dramatically exacerbated by the slightest touch. Eating, speaking and oral care are very painful. Increased salivation, increased regional lymph nodes and pain, a rotting odor from the oral cavity, bleeding gums are observed. If the lesion is localized in the retromolar space in the impaired eruption of 3.8., 4.8., then the complaints are accompanied by trismus - limited mouth opening. The mood is depressed, the skin is pale, covered with small droplets of sweat, the red border of the lips is dry. There is a stinky mouth odor caused by the breakdown of protein from the ulcer tissue with the release of hydrogen sulfide and ammonia, pronounced hypersalivation, the tongue is coated, regional lymph nodes are enlarged, painful, firm to the touch. Gums and oral mucosa are brightly hyperemic, swollen, easily bleeding when touched. First, the tops and then the body of the interdental papillae are covered with necrosis in the form of white-gray, gray-white or gray plaque, which is easily removed by a tampon from the surface of the papillae, after which they look like being cut off (the symptom of cut papillae), and bleeding looks like the dew drop (the symptom of bloody dew). Sometimes spontaneous bleeding is possible. One-sided lesion is characteristic. Ulcerative necrotic lesions are more often localized on the oral mucosa of the cheeks in the

retromolar space, along the line of tooth closure, to the lateral surface of the tongue, on the oral mucosa of the lips and the floor of the oral cavity. In the case of inadequate treatment, acute ulcerative necrotic stomatitis is capable of recurrence and transition to a chronic form. If ulcerative necrotic lesions extend to the palate and tonsils, then Simanovsky-Vincent's quinsy is diagnosed. Unilateral lesions, pain on swallowing, unpleasant mouth odor, and unsanated mouth are characteristic. It can assume 2 forms: ulcerative and pseudo-filmy (diphtheroid). Objectively, one can detect yellowish-white or greyish-dirty plaque on the inflamed and swollen tonsils (diphtheroidal form), after which the ulcer surface up to 5-6 cm with irregular and soft edges is revealed. The bottom is covered with a coating of greenish-gray color, which has a rotting odor. The oral mucosa around the ulcer is hyperemic, swollen. The ulcers can be single and numerous, varying in size and depth. The duration of illness is 2-3 weeks. Diagnostics. Mixed microflora are found in the surface layers, and in the deeper layers there are fusobacteria and spirochetes. Complete blood count changes slightly - a possible increase in leukocytes is characterized by a slight shift to the left, sometimes significant lymphocytosis - up to 70%. ESR - from 15-20 to 30-40 mm / h. In urine, sometimes protein is detected. Treatment. Topical - surgical processing of an infectious wound - removal of necrotized tissues and elimination of local irritants with constant irrigation of the operating field. Antiseptic solutions. In the hydration phase of anesthesia, treatment of the oral cavity and lesions by anaerobic microflora-reducing agents is required. In the dehydration phase, drugs that stimulate reparative processes are used. General therapy - antibiotics, anti-inflammatory drugs (aspirin, butadione), hyposensitizing. Physiotherapy treatment: laser irradiation Prevention. Oral hygiene, regular rehabilitation of the oral cavity and timely treatment of diseases that lead to a decrease in immunity.

## TOPIC 61.

## DIFFERENTIAL DIAGNOSIS OF INFLUENZA ON THE ORAL MUCOSA. DENTIST'S TACTICS.

Flu Differential diagnosis of influenza is carried out with other acute viral diseases (infectious mononucleosis, measles, chicken pox, adenovirus infection, etc.), manifestations of acute leukemia, typhoid fever, leptospirosis. When the course of disease is uncomplicated, the virus disappears from the body from 4 to 5 days, the fever generally lasts up to 4 days, and health is restored in 7-10 days. If the course of the flu is complicated, then the presence of the pathogen is recorded in the body up to 2 weeks. As a consequence sinusitis, otitis, pneumonia can develop. In such cases, the presence of the pathogen in the body is recorded to 2 weeks. Immunity after flu is unstable, since the variability of the immunogenic properties of various types, subtypes and variants of the influenza virus are very high. Treatment. The patient is isolated and prescribed general anti-influenza treatment (detoxification, antiviral, general-strengthening and symptomatic therapy). The dentist recommends oral hygienic care. Local interventions in the presence of changes in the mucous membrane of the oral cavity are determined by only depending on the type of stomatitis (catarrhal, herpetic, aphthous, ulcerativenecrotic, candidal, etc.) and are aimed at preventing the attachment of a secondary infection (the use of local antiseptic agents). Prevention For specific influenza prophylaxis, influenza vaccination is needed. Prophylactic prescription of antiviral drugs is possible during the whole period of the epidemic outbreak.

## TOPIC 62.

## DIFFERENTIAL DIAGNOSIS OF AIDS ON THE ORAL MUCOSA. DENTIST'S TACTICS.

Differential diagnosis. A symptom complex resembling AIDS requires the exclusion of other factors leading to immunodeficiency - occupational, domestic, medical, radiation damage, hematological and oncological diseases, diabetes mellitus, and serious infections. Congenital immunodeficiencies occur from childhood, are characterized by excess in physical and mental development, frequent accession of severe viral, bacterial and fungal infections. Very rarely, a significant decrease in the coefficient of CD4: CD8 and the number of serum immunoglobulins is normal or sharply reduced. Secondary immunological deficiency can occur under the influence of severe inflammatory and oncological diseases, bleeding, radiation, poisoning with certain chemicals, drugs. For diagnosis, the consideration of various harmful factors, immunological (CD4: CD8 ratio) and serological studies are crucial. In the period of primary clinical manifestations, it is necessary to exclude infectious mononucleosis, acute respiratory viral infections, diphtheria of the nasopharynx. Infectious mononucleosis is characterized by leuko- and lymphocytosis with the presence of atypical mononuclear cells, plasma cells. The diagnosis can be confirmed by serological heteroagglutination reactions (Paul-Bunnel, Goff-Bauer), as well as by the increasing titer of antibodies to the Epstein-Barr virus. SARS (severe acute respiratory syndrome) as a rule, linked to similar diseases in other people around the patient and is often linked to the common cold. In a laboratory study, one can identify the corresponding viruses and antibodies to them. Diphtheria is characterized by fibrinous deposits on the tonsils, which extend beyond them. After plaque is removed, the mucous membrane bleeds. Often there is swelling of paratonsillar tissues and cervical tissue. The diagnosis is confirmed by the detection of toxigenic diphtheria bacillus. Diseases of the oral mucosa associated with HIV infection include: various clinical forms of candidiasis; viral infections; hairy (villous) leukoplakia; Kaposi's sarcoma. Diseases that should alert the dentist regarding HIV infection of the patient include squamous cell carcinoma of the oral mucosa, lymphoma. Opportunistic and HIV / AIDS-related infections Herpes simplex Classical course: Acute herpetic gingivostomatitis (primary herpes) and chronic recurrent herpes. The primary elements in herpes simplex are vesicles, after the resolution of which erosions appear in the form of aphthae in primary herpes. Erosions in chronic recurrent herpes have irregular polycyclic outlines. Elements of the lesion are often localized in the hard palate, back of the tongue, gum, cheeks, lips. They are often affected by the red border of the lips and adjacent skin Clinical course in HIV-infected: With HIV infection, herpes simplex often appears without noticeable remissions. Elements of the lesion are located in atypical places. Erosions that occur after opening the vesicles often transform into ulcers up to 3 cm in diameter. The ulcers are crater-shaped, with raised, irregularly shaped edges and a bright red bottom, sometimes ulcers are covered with a grayish-white coating. Ulcers heal slowly, and they are are difficult to treat. Severe forms of recurrent herpes can develop. It affects not only the mucous membrane of the oral cavity, but also the skin. Diagnosis of herpes infection is based on characteristic symptoms, and the diagnosis is confirmed by serological methods or virus isolation. Manifestations in the oral cavity in HIV-infected: Vesicles occur on the mucous membrane of the tongue, soft palate, the bottom of the oral cavity, on the red border of the lips with the transition to the skin. After resolution of the vesicles, erosion often transforms into ulcers with a diameter of 0.5 to 3 cm. The ulcers are crater-shaped, with raised edges, of irregular shape. The bottom of the ulcers is hyperemic, can be covered with a grayish-white coating. Sometimes ulcers resemble lesion elements in multiforme exudative erythema or ulcerative necrotic stomatitis. Without adequate treatment for the manifestations of herpes in the oral cavity, dissemination of the virus into the visceral organs is possible, which aggravates the clinical course and sometimes causes death. Herpes Zoster Classical course: The virus in herpes zoster is

characterized by neurodermatotrophy. In typical cases, the disease manifests itself as vesicular rashes along the nerves. In the oral cavity, the vesicles quickly burst, forming a single or confluent erosion, covered with fibrinous deposits. Crusts form on the skin of the blisters. As a rule, lesions are located on one side of the body behind the course of the intercostal nerves and branches of the trigeminal nerve. Recurrence in this case does not happen. Clinical course in HIV-infected patients: Herpes zoster is a common infection in HIV-infected patients with the usual symptoms. A vesicular rash appears behind the course of the branches of the affected sensory nerve with the development of ganglionatives. Generalized skin changes occur in 2-4% of cases of recurrence of herpes zoster or its dissemination indicates progression of HIV infection. Patients with suspected herpes zoster or with a confirmed diagnosis of younger 60 years should be tested for HIV / AIDS Manifestations in the oral cavity in HIV-infected patients: Oral mucous membrane is rarely affected. Some patients have facial paralysis and trigeminal neuralgia. Sarcoma Kaposi Classical course: Patients with HIV infection often develop Kaposi's sarcoma, malignant lymphomas, sometimes squamous cell carcinoma of the tongue and anorectal region, glioma. Kaposi's sarcoma is a malignant tumor of lymphatic vessels, is found mainly in people older than 60 years, often in men. In Kaposi's sarcoma is not affiliated the HIV, there are spots, nodules or plaques brown-red or bluish-red color mostly on the skin of the lower extremities. The disease progresses slowly and leads to the death of the patient in 10-15 years. With HIV infection, Kaposi's sarcoma occurs at the stage of secondary diseases. The etiological role is played by human herpes simplex virus type 8. Clinical course in HIV-infected: With HIV infection, Kaposi's sarcoma develops in young people. The disorder is multiple, asymmetric, widespread. The disease tends to rapidly generalize with damage to the mucous membranes of the oral cavity, upper respiratory tract, internal organs, and lymph nodes, and is often associated with opportunistic infections. Perhaps an isolated lesion of the mucous membranes and lymph nodes. In half of the cases, lymphadenopathy is associated with Kaposi's sarcoma metastasis. Mortality is high among such patients. Manifestations in the oral cavity in HIV-infected people: In the oral cavity in HIV-infected patients, Kaposi's sarcoma appears as bluish, red, cherry-violet or black spots. Sometimes the elements of the lesion are not pigmented. Flat spots in the initial stages, then enlarge, split into parts and ulcerate. At the stage of ulceration, the elements of the lesion are sharply painful. Ulcers are not susceptible to epithelization. Most often, Kaposi's sarcoma in the oral cavity is localized on a hard and soft palate, less commonly found on the gums, tongue. Kaposi's sarcoma on the gum may resemble epulis. In the tongue, lesion elements are located, as a rule, in the area of the pinciform papillae. Hairy Leukoplakia Classical course: HIV infection does not occur ouside. The clinical course of HIV-infected patients: Hairy leukoplakia (oral viral leukoplakia, flat condyloma, fleecy leukoplakia) occurs as a result of the passage of the Epstein-Barr virus into the epithelial cells of the oral mucosa. With the appearance of hairy leukoplakia, the possibility of developing AIDS increases. It occurs in 98% of AIDS patients. Only the mucous membrane of the oral cavity is affected. Manifestations in the oral cavity in HIV-infected patients: Clinically, hairy leukoplakia is manifested by damage to the lateral surfaces and back of the tongue. Foci of leukoplakia can spread to the mucous membrane of the cheeks, the bottom of the oral cavity, and the sky. Elements of the lesion are areas of hyperkeratosis in the form of whitish lines, folds, protrusions, craters. Areas of hyperkeratosis are not removed, they look like flat plaques, similar lesions in classical leukoplakia. The surface of the plaques can be smooth and rough. Differential diagnosis of hairy leukoplakia is carried out with contact allergic reactions to dental materials, true leukoplakia and leukoplakia of smokers, lichen planus, hyperplastic candidiasis, damage to the oral mucosa with galvanic effects. Candidiasis Classical course: Candidiasis is caused by opportunistic fungi of the genus Candida, which are representatives of the normal microflora of the oral cavity, mucous membrane of the gastrointestinal tract, and skin. Acute pseudomembranous candidiasis (thrush) develops in the oral cavity with curdled, easily swollen deposits on the hyperemic mucous membrane. Acute atrophic candidiasis is accompanied by severe

hyperemia of the mucous membrane, plaque is absent or persists in deep folds. In chronic hyperplastic candidiasis, a thick layer of a tightly sitting plaque in the form of nodules or plaques tightly soldered to the surrounding mucous membrane forms on the hyperemic oral mucosa. Clinical course in HIV-infected: Mycoses with HIV infection often occur, these are opportunistic and AIDS-associated diseases. The development of mycoses always indicates the progression of HIV infection and is usually observed during the transition of the disease to the stage of secondary diseases. Often there are cryptococcosis, histoplasmosis and coccidioidomycosis, zygomycosis, penicillinosis, candidiasis. In HIV-infected patients, the clinical manifestations of candidiasis depend on the severity of immunodeficiency. With the oropharyngeal form, the mucous membrane of the oral cavity is involved in the process with a clinic of candidal tonsillitis, stomatitis. In severe forms of immunodeficiency, when the number of CD4 + cells falls below 200 in 1 μl, the process from the mucous membrane of the oral cavity drops down and affects the esophagus, trachea, bronchi, and lungs. Manifestations in the oral cavity in HIV-infected people: Most often, prolonged acute pseudomembranous candidiasis occurs. Clinical manifestations are identical to ordinary candidiasis - white curdled plaque on the mucous membrane. Plaque is easy to remove. Under it, the hyperemic mucous membrane is exposed. Acute atrophic candidiasis may also develop in the form of sites of atrophy of the filiform papillae of the tongue. Hyperplastic candidiasis is rare. In the differential diagnosis of candidiasis, the symptoms of similar diseases should be taken into account: leukoplakia; lichen planus; allergic stomatitis; traumatic lesions. Diagnosis of candidiasis is based on typical clinical signs and the results of a bacterioscopic examination of scraping from the affected oral mucosa. The presence of a multiple fungus of the genus Candida in the form of spores and hyphae always confirms the diagnosis of candidiasis. Seborrheic dermatitis Classical course: It is not found outside of HIV infection Clinical course in HIV-infected: In HIV-infected, seborrheic dermatitis has the appearance of areas of mild erythema on the skin of the face, neck, scalp, behind the auricles, on the extensor surfaces of the hands. Typical lesions are eyebrows, nose, hairline, lip surfaces, nasolabial folds. Erythematous areas are not sharply limited from apparently healthy skin, have irregular shapes and are covered with a small amount of thick scales and yellow crusts. Small vesicles with serous or purulent contents may occur. After resolving the elements of the lesion, foci of atrophy remain. Patients complain of severe itching, the spread of seborrheic dermatitis to all skin indicates a positive prognosis of the disease. Manifestations in the oral cavity in HIV-infected patients: The mucous membrane of the oral cavity is not affected.

## TOPIC 63.

## DIFFERENTIAL DIAGNOSTICS OF MANIFESTATIONS OF MURRAIN, INFECTIOUS MONONUCLEOSIS. DENTIST'S TACTICS.

Fever

Fever is rarely the sole manifestation of Epstein-Barr virus (EBV) infectious mononucleosis. Because most patients with EBV infectious mononucleosis usually have fever, pharyngitis, and lymphadenopathy, the differential diagnoses are those of an infectious mononucleosis–like illness, which include infectious mononucleosis due to cytomegalovirus (CMV), human herpesvirus 6 (HHV-6), acute HIV disease, toxoplasmosis, and anicteric viral hepatitis.

These infectious diseases, which have presentations similar to those of infectious mononucleosis, have also been termed heterophile-negative infectious mononucleosis because the heterophile test and EBV serology findings are negative in these patients. In rare cases, EBV infection has been reported as a cause of fever of unknown origin (FUO).

Fevers due to EBV infectious mononucleosis may reach 103-104°F but are usually less than 102°F. Relative bradycardia is a rare finding in patients with EBV mononucleosis and suggests myocardial involvement (eg, myocarditis). Persistent fever or a recrudescence of fever after clinical recovery should suggest an alternate diagnosis.

Pharyngitis

Pharyngitis is one of the cardinal manifestations of EBV infectious mononucleosis. Exudative pharyngitis may resemble streptococcal pharyngitis. Patients with EBV mononucleosis may present with a pseudomembrane resembling Corynebacterium haemolyticum or Corynebacterium diphtheriae. However, these infections do not have the associated findings that comprise the infectious mononucleosis syndrome and should present no diagnostic difficulties.

Palatal petechiae are most commonly found in association with EBV infectious mononucleosis but may also be observed in group A streptococcal pharyngitis. In patients with pharyngitis, palatal petechiae may also be a sign of a granulocytosis caused by aplastic anemia or a lymphoreticular malignancy involving the bone marrow (eg, acute leukemias or lymphomas).

Uvular edema is an important and fairly specific finding in individuals with EBV infectious mononucleosis. The causes of heterophile-negative infectious mononucleosis and group A streptococcal pharyngitis are not accompanied by uvular edema. Although uncommon, uvular edema has important diagnostic significance when present. Patients with a C1q deficiency may present with uvular edema; however, these patients have no evidence of pharyngitis, fever, or adenopathy and should not be confused with patients with EBV infectious mononucleosis.

The posterior oropharynx in patients with EBV infectious mononucleosis is uniformly erythematous. This is in contrast to the discreet pretonsillar purplish discoloration observed in chronic fatigue syndrome (CFS) that has been termed "crimson crescents." Crimson crescents, a possible marker of CFS, occur in the absence of surrounding posterior pharyngeal erythema. Patients with CFS do not present predominantly with pharyngitis.

Patients with heterophile-negative infectious mononucleosis have minimal or mild nonexudative pharyngitis. Palatal petechiae and uvular edema are usually absent, and exudative pharyngitis is not a feature of these infectious diseases.

Lymphadenopathy

Any or all chains may be enlarged in individuals with EBV infectious mononucleosis. Lymphadenopathy is always bilateral and symmetrical in all patients, including those presenting with generalized adenopathy. Bilateral posterior cervical adenopathy is most highly suggestive of EBV infectious mononucleosis.

Some of the causes of heterophile-negative infectious mononucleosis may manifest as bilateral posterior cervical adenopathy (eg, rubella), but other signs and symptoms serve to differentiate these patients from those with EBV infectious mononucleosis. Patients with rubella have other associated findings, including the distribution and progression of the rash and occipital or preauricular adenopathy; usually, they do not have generalized adenopathy, and liver involvement is not a feature of rubella infections.

Acquired toxoplasmosis in adults has minimal pharyngeal or hepatic involvement, but adenopathy may be prominent. In contrast to EBV infectious mononucleosis, generalized adenopathy is not a feature of toxoplasmosis. Highly characteristic of toxoplasmosis is asymmetrical lymphadenopathy limited to an isolated lymph node group. Patients with toxoplasmosis have little or no fever, fatigue, or pharyngitis, which helps differentiate toxoplasmosis-induced infectious mononucleosis from EBV-induced infectious mononucleosis.

Patients with HHV-6 infection may have a presentation that is identical to that of infectious mononucleosis, but fatigue is usually less prominent. Isolated posterior cervical adenopathy may also occur with in HHV-6 infectious mononucleosis.

Patients with HIV infection with acute seroconversion may present with a mononucleosislike illness with a maculopapular rash, mild pharyngitis, and lymphadenopathy. The adenopathy in HIV may be localized or generalized, but splenomegaly is not a feature of uncomplicated early HIV infection. Adenopathy localized to a lymph node group in a patient with HIV infection should suggest a lymphoma rather than a primary manifestation of acute HIV infection.

Anicteric hepatitis is rarely, if ever, accompanied by localized or generalized adenopathy. The finding of bilateral posterior cervical adenopathy argues against the diagnosis of anicteric hepatitis in a patient with otherwise unexplained fatigue.

CMV mononucleosis is the heterophile-negative cause of infectious mononucleosis that is most likely to be confused with EBV infectious mononucleosis. CMV infectious mononucleosis may be indistinguishable in clinical presentation from EBV but is usually not accompanied by posterior cervical adenopathy. Nonexudative pharyngitis is minimal or absent, and splenomegaly is less common than in EBV infectious mononucleosis. CMV infectious mononucleosis is characterized by its prolonged course and prominent liver involvement. Serum transaminases may persistently remain mildly to moderately elevated for prolonged periods. In patients presenting with infectious mononucleosis that has persisted for 6-12 months after a mononucleosislike illness, the condition is most likely due to CMV infectious mononucleosis.

Pseudolymphoma

Patients receiving certain drugs, particularly phenytoin (Dilantin), may present with a mononucleosislike illness. Such patients usually present with fever and generalized adenopathy without pharyngitis or liver involvement. The finding of isolated groups of lymph node enlargement (eg, posterior cervical adenopathy) argues against the diagnosis of drug-induced pseudolymphoma.

Atypical lymphocytes may be present in patients with drug fevers and pseudolymphomas, but the percentage of atypical lymphocytes is less than 10%, in contrast to EBV-induced infectious

mononucleosis. Pseudolymphoma may be confused with lymphomas but may be differentiated readily based on a lack of eosinophils or basophils, which may be present in the peripheral smear of patients with lymphoma, or the finding of abnormal lymphocytes in the peripheral smear versus the atypical lymphocytes of pseudolymphoma and viral infections, which are reactive and atypical but not abnormal.

Anicteric hepatitis

Patients with anicteric hepatitis present with anorexia, malaise, and fatigue. Pharyngitis may occur, but it is mild and nonexudative. Generalized adenopathy and splenomegaly may occur with anicteric hepatitis, but this occurs much more infrequently than with EBV infectious mononucleosis. Anicteric hepatitis is most likely to be confused with EBV infectious mononucleosis in elderly individuals who present with hepatitis. Positive findings on hepatitis serology and negative findings on heterophile/EBV serology differentiate these two infectious diseases.

Splenomegaly

Splenomegaly may be classified according to the degree of splenic enlargement and whether it occurs alone or as part of generalized lymph node involvement. Although, in rare cases, splenic rupture is the initial clinical manifestation of EBV infectious mononucleosis, the splenomegaly of EBV infectious mononucleosis is usually accompanied by localized or generalized adenopathy. In the absence of splenic rupture, patients with EBV infectious mononucleosis do not present with isolated splenomegaly in the absence of other findings. The many systemic disorders that manifest as splenomegaly in the absence of lymphadenopathy, eg, brucellosis, lymphoma, and subacute bacterial endocarditis (SBE), are readily differentiated from EBV infectious mononucleosis with splenic enlargement.

Generalized adenopathy may occur with many infectious and noninfectious diseases, most commonly group A streptococcal infections, systemic lupus erythematosus (SLE), and sarcoidosis. Because the spleen is part of the RES, most cases of generalized adenopathy are accompanied by splenomegaly. However, most disorders with presentations that predominantly involve generalized adenopathy rarely involve splenomegaly, and, when present, the splenic enlargement is not prominent (eg, generalized adenopathy is common in SLE, but splenomegaly is uncommon). Generalized adenopathy with prominent splenomegaly should suggest EBV infectious mononucleosis. A diagnosis of EBV infectious mononucleosis in the absence of bilateral posterior cervical adenopathy with or without generalized adenopathy or splenomegaly should raise suspicion of the diagnosis.

Leukocytosis

Most patients with EBV infectious mononucleosis have a mildly to moderately increased peripheral WBC count, usually in the range of 12-20,000 cells/µL. Leukocytosis is a nonspecific finding in medicine in general and in infectious disease in particular. Leukocytosis has importance in ruling out some other causes of heterophile-negative infectious mononucleosis. Leukopenia, rather than leukocytosis, is expected in patients with CMV, rubella, HHV-6, acute HIV, and anicteric hepatitis-related infectious mononucleosis. Patients with toxoplasmosis and pseudolymphoma usually have a normal rather than an elevated peripheral WBC count.

Lymphocytosis

Lymphocytosis is one of the classic hematological abnormalities associated with EBV infectious mononucleosis. Relative lymphocytosis (≥ 60%) plus atypical lymphocytosis (≥ 10%) are

the characteristic findings of EBV infectious mononucleosis. The causes of heterophile-negative infectious mononucleosis rarely, if ever, have a relative lymphocytosis in excess of 60%. However, in contrast, atypical lymphocytosis is a common feature of any agent responsible for heterophile-negative infectious mononucleosis. The important differential diagnostic point is that the atypical lymphocytosis of EBV infectious mononucleosis is not simply equal to or greater than 10% but is frequently equal to or greater than 30%. An important point is that EBV infectious mononucleosis is more likely to be the cause of atypical lymphocytosis in patients with infectious mononucleosis with greater degrees of atypical lymphocytosis.

Thrombocytopenia

Mild transient thrombocytopenia is not uncommon in EBV infectious mononucleosis. Severe or persistent thrombocytopenia should suggest an alternate diagnosis, eg, acute HIV or other viral infectious diseases. Thrombocytosis is not a feature of EBV infectious mononucleosis, and its presence should suggest an alternate diagnosis, eg, malignancy due to lymphoma in adults or, in children, Kawasaki disease.

Increased serum transaminases

An early, transient, mild increase in serum transaminases is characteristic of EBV infectious mononucleosis. High elevation of the serum transaminases should suggest viral or drug-induced hepatitis. The mild elevations of serum transaminases that occur in infectious mononucleosis are useful diagnostic tests before the heterophile becomes positive. Mild-to-moderate elevations of the serum transaminases that persist over months in a patient with a mononucleosislike illness should suggest CMV rather than EBV infectious mononucleosis.

Erythrocyte sedimentation rate

Erythrocyte sedimentation rate (ESR) elevations occur in virtually all patients early in the course of EBV infectious mononucleosis. Similar to the early and mild elevations of the serum transaminases that occur in EBV infectious mononucleosis, an elevated ESR can be a useful diagnostic test early in the course of the disease in patients presenting with pharyngitis. While the ESR is elevated in patients with EBV as well as with other causes of viral pharyngitis, it is not elevated in patients with group A streptococcal pharyngitis. In patients with pharyngitis, elevations of the ESR are most useful in differentiating EBV infectious mononucleosis from group A streptococcal pharyngitis early in the course of the disease before the heterophile or the antistreptolysin-O (ASO) titers increase.

Maculopapular rash

Maculopapular rash may be caused by a large variety of infectious and noninfectious agents. Maculopapular rashes associated with pruritus are not caused by infectious agents. Nonpruritic maculopapular rashes may be caused by a wide variety of infectious and noninfectious disorders. The differential diagnoses of rash and fever depend largely on the distribution of the rash. Unfortunately, maculopapular rashes are generalized, offering little opportunity to narrow differential diagnostic possibilities. Therefore, the best approach to the differential diagnoses of maculopapular rashes must depend on their clinical behavior, rate of progression and/or recession, and associated nondermatologic features.

The rash of EBV infectious mononucleosis occurs in the first few days and is transient, mild, and evanescent. The early rash of EBV infectious mononucleosis is easily missed by patients and physicians. The causes of heterophile-negative infectious mononucleosis are usually

unaccompanied by a rash, except for acute HIV infection, which has a rash indistinguishable from EBV primary infection.

Rubella is the least likely exanthem to be confused with EBV mononucleosis; the rash persists longer and is not accompanied by the other features that are characteristic of infectious mononucleosis, eg, prominent pharyngitis. Patients with measles have conjunctival injection, coryza, and a rash that is maculopapular but blotchy and progresses from the head downward, differentiating it from the rash of EBV. A rash caused by contact dermatitis or drug-induced maculopapular rashes are pruritic, differentiating them easily from the rash of EBV. Erythrodermas with an initial presentation of maculopapular rashes caused by systemic disorders are usually persistent (eg, Sézary syndrome), in contrast to the evanescent mild rash of EBV infectious mononucleosis.

Periorbital edema

Periorbital edema is caused by various agents. Periorbital edema is an uncommon, and therefore fairly specific, physical finding in infectious diseases. Bilateral periorbital edema not associated with generalized edema, eg, nephrotic syndrome, should suggest trichinosis, Kawasaki disease, allergic reactions, or bilateral periorbital cellulitis. Unilateral periorbital edema suggests conditions such as thyrotoxicosis, retro-orbital eye tumor, Chagas disease, insect sting, and unilateral conjunctivitis. EBV infectious mononucleosis is characterized by early and transient bilateral upper-lid edema.

In contrast to the disorders mentioned above, which are either unilateral or bilateral and involve the periorbital area, with or without the eyelids, the external eye involvement of EBV infectious mononucleosis is characterized by bilateral upper-lid edema. This finding first was described by Hoagland and is referred to as Hoagland sign (see Physical). In contrast, infectious mononucleosis is characterized by palpebral edema rather than periorbital edema.

Splenic rupture

Splenic rupture is a rare complication of EBV infectious mononucleosis. Splenic rupture may be the presenting sign of EBV primary infection.

Meningoencephalitis

Meningoencephalitis is a very rare manifestation of EBV infectious mononucleosis. Patients who have unusual neurologic manifestations (eg, scalp tenderness, optic neuritis) usually have other features of EBV infectious mononucleosis, which should suggest the cause of the patient's neurologic symptoms. Neurologic manifestations as the sole indication of EBV infectious mononucleosis are rare. The diagnosis of EBV infectious mononucleosis is a syndromic diagnosis, which is based on the association of fever, pharyngitis, and lymphadenopathy in conjunction with the characteristic hematologic abnormalities of EBV infectious mononucleosis. The clinician should look for associated features of infectious mononucleosis to rule in or rule out the possibility in patients with otherwise unexplained mental status changes.

Chronic fatigue

Profound initial fatigue and malaise is a feature of EBV infectious mononucleosis. Fatigue has extensive differential diagnoses because many systemic disorders are accompanied by fatigue. The cause of fatigue in the patient with EBV infectious mononucleosis is suggested by the constellation of signs, symptoms, and laboratory abnormalities that suggest the diagnosis. In the absence of such findings, other causes of fatigue should be sought.

Many infectious agents, including EBV infectious mononucleosis, are known to initiate a state of chronic fatigue. Appreciate that EBV may trigger chronic fatigue, but it does not cause chronic fatigue. The fatigue of EBV infection usually resolves within 3 months and uncommonly lasts for longer than 6 months. Patients with CFS have otherwise unexplained fatigue for a duration equal to or greater than 1 year (for a full discussion on CFS, see Chronic Fatigue Syndrome). In summary, acute, but not chronic, fatigue is a feature of EBV infectious mononucleosis.

Chronic infectious mononucleosis is rare and occurs in those with immunologic abnormalities. Such patients present with fever, lymphadenopathy, persistently elevated serum transaminases, and pancytopenia. Eye or neurologic abnormalities may also be present. Importantly, patients with CFS have none of these findings. Patients with acute EBV infection do not have pancytopenia, and their clinical presentation rapidly resolves. Chronic infectious mononucleosis is a diagnosis that should be made rarely and carefully. Commonly, patients and physicians equate increased EBV immunoglobulin G (IgG) VCA antibody titers with chronic infectious mononucleosis or CFS because more than 90% of the population has increased EBV IgG VCA antibodies. The associated findings of fatigue are coincidental and are not related causally.

## TOPIC 64.

## DIFFERENTIAL DIAGNOSIS OF MANIFESTATIONS OF DIPHTHERIA ON THE MUCOUS MEMBRANE OF THE ORAL CAVITY. DENTIST'S TACTICS.

Diphtheria is an acute infectious disease that is transmitted by airborne droplets. The causative agent is Leffler's diphtheria bacillus, the pathogenic properties of which are determined by its exotoxin. Clinically the incubation period is 2-10 days. Then there is a sore throat, t38-390 C, general weakness, heart pain, lack of appetite. From the first hours of the disease, hyperemia and swelling of the mucous membrane of the tonsils (diphtheria angina) develops. Then massive fibrinous films of white or grayish-white color are formed, which extend to the mucous membrane of the nasal part of the pharynx, the hard palate and can spread to the gums, mucous membrane of the cheeks, tongue. The film coating is tightly soldered to the underlying tissues, has a sweetish smell and is very difficult to remove, exposing the bleeding surface. The occurrence of films is associated with a fibrinous form of inflammation and is a local reaction to the deepening of Leffler's bacillus and its toxins. Diagnosis of the disease is based on data from a bacteriological study, conducting a passive hemagglutination reaction. Differential diagnosis - manifestations of scarlet fever, infectious mononucleosis, Simanovsky-Vincent sore throat, acute herpetic stomatitis, erythema multiforme exudative, acute candidiasis, leukemia. Treatment of patients is carried out in a hospital! infectious diseases hospital, and consists in the introduction of diphtheria antitoxin, anti-inflammatory drugs, vitamins, heart drugs. Locally use antiseptics, antibiotics, enzymes, painkillers and keratoplastic agents. For the prevention of diphtheria, toxoid vaccinations are important.

## TOPIC 65.

## DIFFERENTIAL DIAGNOSIS OF MANIFESTATIONS OF TUBERCULOSIS ON THE ORAL MUCOSA. DENTIST'S TACTICS.

Tuberculosis is a chronic infectious disease caused by mycobacterium tuberculosis (Koch's bacillus). It enters the mucous membrane of the mouth by the hematogenous, lymphogenous or exogenous route. In the oral mucosa, tuberculosis occurs in the form of a secondary lesion and manifests itself in the form of: lupus erythematosus tuberculosis, miliary ulcerative tuberculosis, colic tuberculosis (scrofuloderma). Tuberculosis lupus. The main element of the lesion is lupoma, a specific tuberculous tubercle (tuberculum), red or yellow-red, soft consistency with a diameter of 1-3 mm. Lupomas are located in groups, fresh ones are formed on the periphery, and those in the center are prone to decay, after which ulcers with soft, uneven, saped edges, swollen and less painful form. The bottom of the ulcer is covered with yellow-red raspberry-like growths that bleed easily. Stages of the process: infiltrative, hilly, ulcerative and cicatricial. Localization of the elements of the lesion: red border of the upper lip, gums and alveolar process of the upper jaw in the region of the front teeth and fangs. Sometimes the process moves to a hard and soft palate. Regional lymph nodes are enlarged, dense, bundled. Diagnosis: a symptom of "apple jelly" during dioscopy and a symptom of a probe failing in a lupoma (Pospelov phenomenon). The reaction of Pirke is positive. Histological examination reveals epithelioid cells, giant Pirogov-Lanhgans cells and peripheral lymphocytes. Differential diagnosis: manifestations of tertiary syphilis (tubercle syphilis), leprosy, lupus erythematosus. Miliary - ulcerative tuberculosis. It occurs in patients with severe forms of pulmonary tuberculosis or larynx. Mycobacterium tuberculosis with the sputum of the patient settles in the places of the oral mucosa, which is prone to injuries (back of the tongue, mucous membrane of the cheeks along the line of closure of the teeth, soft palate, clear). The microflora multiplies, and typical tuberculous tubercles arise, which decay in the center and form shallow ulcers, which are creeping in nature, with uneven soft undercut edges. The bottom and edges of the ulcer are granular (due to tubercles), covered with a yellow-gray coating. Small abscesses (Trill grains) are determined. Inflammation around the ulcer is weak. Lymph nodes are enlarged, tight - elastic, painful. Diagnosis: the general condition of the patients is important (weight loss, excessive sweating, shortness of breath, fever) elevated ESR in the blood, leukocytosis, lymphocytosis. In scrapings from ulcers, Pirogov-Lanhgans cells are found, with bacteriological examination - Koch bacilli. Differential diagnosis: performed with a gummy ulcer in syphilis, Vincent’s necrotic stomatitis, radio mucositis, a traumatic chronic ulcer, trophic, cancer ulcer and manifestations of secondary syphilis. 5 Colquatic tuberculosis (scrofuloderma) a form of secondary tuberculosis. The main element of the lesion is the node that forms in the deep layers of the mucous membrane. 3 time, the nodes disintegrate and ulcers of irregular shape, soft consistency arise, with eaten sap edges and sluggish granulations at the bottom. Ulcers are slightly painful; uneven shaggy scars form during healing. Differential diagnosis: gummous, cancerous, trophic ulcers, Seton's stomatitis, actinomycosis. Tactics of the dentist: if the dentist diagnosed the patient with tuberculosis, he should send him for a consultation with a TB doctor. When confirming the diagnosis, treatment is carried out in a tuberculosis dispensary.

## TOPIC 66.

## DIFFERENTIAL DIAGNOSIS OF MANIFESTATIONS OF SYPHILIS, GONORRHEA. DENTIST'S TACTICS.

Syphilis is a chronic infectious disease caused by a pale spirochete (spirochete palida). In the oral cavity, it manifests itself in all stages of the disease: primary, secondary and tertiary syphilis. Primary syphilis is a hard chancre that can be localized in various parts of the oral cavity, mainly on the lips, tongue, and corners of the mouth. The incubation period lasts 14-20 days. Clinically. First, erosion of a bright red color occurs, then a defect in the form of an ulcer. An infiltrate forms around the lesion. The edges of the ulcer are raised, roll-shaped, in connection with which the solid chancre rises above the level of the mucous membrane. On palpation, a painless cartilage seal is felt. Regional lymph nodes are dense, mobile, painless. Diagnostics. When bacteriological examination of the lesion is found pale spirochete. Differential diagnosis: tuberculous ulcer, trophic, decubital, cancer ulcer. Secondary syphilis in the oral cavity manifests itself in the form of individual roseola, or erythema (roseolous syphilis), or papules (papular syphilis) and less often pustules - pustular syphilis. Diagnosis of secondary syphilis is confirmed by the presence of pale treponema in the lesions and positive serological reactions. Differential diagnosis: lichen planus, leukoplakia, allergic stomatitis. Tertiary syphilis manifests itself in the oral cavity: in the form of gum, tubercular syphilis, sclerosed glossitis. Gumma is a node that clearly protrudes above the level of the oral mucosa the size of a bean, red in color with a dense consistency. Gradually, the color gains a bluish tint, an infiltrate is created, which turns into bone necrosis of the hard palate, a sequestration develops, which exfoliates and a message forms between the oral cavity and the nose. If the gumma on the tongue, then it is laid in the linguistic muscles. When it decays, an ulcer forms, which has inclined and dense edges. The ulcers are deep, painless, and crater-like, with a dirty gray bottom. With the reverse development, they heal with the formation of deep retracted scars. 6 Differential diagnosis: with cancer, tuberculous ulcers and decubital. Sclerosing glossitis from the folded tongue. Treatment of patients is carried out in a dermatovenereological clinic

Gonorrhea is an acute infectious disease of the oral mucosa caused by gonococcus. The incubation period is from 1 day to 1 month. After 3-4 days, gonococci that enter the oral mucosa reach the subepithelial layer of connective tissue through the intercellular spaces and cause an inflammatory reaction with the formation of purulent exudate, considered as migration of neutrophils and plasmocytes to the pathogen invasion site. Complaints of patients are absent. The mucous membrane of the lips, gums, lateral and lower surface of the tongue and the bottom of the oral cavity, pharynx, tonsils, larynx is brightly hyperemic and covered with a dirty - gray, sometimes greenish purulent coating with an unpleasant odor. Often, unilateral arthritis of the jaw-temporal joint develops, characterized by significant pain, then acute inflammation of the joint develops, swelling appears, the skin in the joint area turns red; it becomes tense and sharply painful. Diagnosis is confirmed by the presence of gonococcus with exudate microscopy. 7 Differential diagnosis is carried out with diphtheria, drug allergic stomatitis, erythema multiforme exudative, ulcerative stomatitis, fungal stomatitis. General and local treatment consists in taking antibiotics (ceftriaxone, cefazolin, cefabid, ciprinol) for 1-2 weeks. Topically applied enzymes, antiseptic irrigation. The main therapy is carried out by a dermatovenerologist.

## TOPIC 67.

## DIFFERENTIAL DIAGNOSIS OF LESIONS OF THE MUCOUS MEMBRANE OF THE ORAL CAVITY IN DISEASES OF THE DIGESTIVE CANAL. MODERN METHODS OF TREATMENT AND PREVENTION.

Changes in the oral mucosa at diseases of internal organs and systems
Changes in the oral mucosa at diseases of gastrointestinal tract
Changes in the tongue.
They are the most typical at diseases of gastroin- testinal tract. Among these signs the furred tongue is most frequently revealed. The disturbance of the process of cornification and destruction of epithelial cells in the tongue papillae, as a result of the neurotrophic disorders, play an important role in the formation of fur on the tongue. The nature of food, the composition of microflora, the oral hygiene influence the accumulation of fur. If the epithe- lium detachment is not observed, the keratinized cells remain, dead leukocytes and microorganisms of saliva are joined and form fur. In this case the favorable conditions for multiplication of microorganisms are created, in particular, a sig- nificant amount of fungi is formed. All of this is the basis of fur. Fur is revealed with gastritis, stomach ulcer and duodenal ulcer, new formations in the stomach and other illnesses. Edema of the tongue is the second sign of gastrointestinal diseases. It does not cause subjective sensations in patient. Edema of the tongue is observed in case of chronic bowel diseases, which is explained by the disturbance of the sucking ability in the gut and gut barrier function. The changes in the different groups of the papillae of the tongue are observed at diseases of gastrointestinal tract. Depending on their state hyper- and hypoplastic glossitis aredistinguished.

Hyperplastic glossitis is observed in patients in case of gastritis with increased acidity. It is accompanied by the hypertrophy of papillae, presence of fur, a slight increase in size of the tongue as a result of edema.
Hypoplastic glossitis is characterized by the atrophy of papillae, sometimes sharply expressed, in consequence of which it become varnished, with bright spots and strips. The atrophy of the tongue papillae causes the sensation of burning, tin- gling, pain when eating. Such changes in the tongue papillae are observed in case of gastritis with lowered secretion, ulcer, gastroenteritis, biliary tract disease.

## TOPIC 68.

## DIFFERENTIAL DIAGNOSTICS OF LESIONS OF THE MUCOUS MEMBRANE OF THE ORAL CAVITY IN DISEASES OF THE CARDIOVASCULAR SYSTEM. MODERN METHODS OF TREATMENT AND PREVENTION.

Lesions of the oral mucosa at cardiovascular pathology

Changes of oral mucosa at cardiovascular diseases depend on the degree of insufficiency of blood circulation, state of vascular wall, etc.

Cyanosis of oral mucosa, lips and the tongue of bright red or crimson color are characteristic for the acute period of myocardial infarction.

The appearance of trophic changes of oral mucosa, up to the formation of ulcers, is observed predominantly in patients with the decompensated defects of heart and the disturbance of blood circulation of III, sometimes II degree.

Trophic ulcers are localized predominantly in the rear of the mouth, on the cheek mucosa, alveolar branch, on the tongue, on the mucous a pear-shaped area, etc. Ulcers have different sizes (2-5 mm and more). They are covered with pale gray fur with the fetid smell; it is sharply painful when touching and eating. The inflammatory reaction in the surrounding tissues is absent. The treatment of such changes of oral mucosa provides for the liquidation of the insufficiency of blood circulation in combination with the local symptomatic therapy.

## TOPIC 69.

## DIFFERENTIAL DIAGNOSIS OF LESIONS OF THE MUCOUS MEMBRANE OF THE ORAL CAVITY IN DISEASES OF THE ENDOCRINE AND NERVOUS SYSTEMS. MODERN METHODS OF TREATMENT AND PREVENTION.

Changes in the oral mucosa at endocrine diseases Diabetes mellitus.

The most characteristic changes in the oral cavity are xero- stomia, catarrhal stomatitis and glossitis, fungal stomatitis, mycotic perleche, paresthesias of oral mucosa, trophic disorders, Lichen ruber planus. Dentist conducts the treatment of a patient together with the endocrinologist.Symptomatic treatment is prescribed with the expressed changes in the oral cavity taking into account their manifestation.

Myxedema is developed with the insufficiency of the function of the thyroid gland. The face of patient takes the unique form: lips and nose are thickened, upper The eyelids are sharply edematic, and facial expression is indifferent. In patients ane- mia, edema and dryness of oral mucosa are observed. Myxedema is accompanied by a marked increase of the tongue, which is sometimes not placed in the oral cav- ity, an increase of the lips, gums. Dentist conducts the sanitation of the oral cavity and symptomatic treatment if necessary. Pregnancy gingivitis is an inflammation of gums, which first appears dur- ingpregnancy or is exacerbated by pregnancy. The development of the disease is connected with restoring of hormonal balance during this period. In the first half of pregnancy the catarrhal gingivitis is noted. In the second half - the course of dis- ease is heavy, with the development of proliferating process in the gums. In the initial stage of gingivitis the gingival edge becomes clear red, swells, bleeds easily. Gradually the affected gum becomes dark red, cyanotic, increases and with the pres- ence of local stimuli the hypertrophic gingivitis develops. Hypertrophic gingivitis in pregnancy tends to polypous growth of separate papillae. Sometimes false epulis develop. The hypertrophied gum covers the entire dental crown, bleeds easily. Treatment. The local treatment of pregnancy gingivitis is conducted through the principles of the treatment of catarrhal or hypertrophic gingivitis.

Itsenko-Cushing disease. The oral mucosa in such patients is edematic, the imprints of teeth on the tongue and the cheeks are observed. Appearing trophic disorders lead to the appearance of erosions and ulcers which are characterized by a prolonged course. Candidiasis is frequently observed.

## TOPIC 70.

## DIFFERENTIAL DIAGNOSTICS OF MANIFESTATIONS OF BLOOD DISEASES AND ORGANS OF HEMATOPOIESIS ON ORAL MUCOSA. LEUKEMIA, AGRANULOCYTOSIS. DENTIST'S TACTICS.

Leucosis is the malignant disease of the hemopoietic organs, which appears as a result of progressive cellular hyperplasia in the hemopoietic organs, when the processes of cellular division (proliferation) prevail above the processes of ripening (differentiation). Pathomorphological substratum of disease is leukemia blast cells which correspond to primordial elements of one of the hematopoietic lineage.

The clinical picture of acute leucosis determines 4 leading syndromes: hem- orrhagic, hyperplastic, anaemic and intoxicating. Hemorrhagic syndrome is manifested in the form of petechiae, ecchymosis, hematomas on the skin and mucous membrane, or the profuse bleedings. In the oral cavity the most characteristic are: acute bleeding of gums, pres- ence of hemorrhages on mucous membrane of checks along the line of the join- ing of teeth, in the tongue, the palate. Severe hemorrhages and hematomas some- times are revealed. Hyperplastic processes are manifested in the increase of the lymph nodes, liver, spleen, tonsils. Frequently hyperplasia is combined with ul- ceronecrotic changes in the gums. Furthermore, necroses are revealed also in the tonsils, the pear-shaped area and other parts of oral mucosa. Its tendency toward the propagation in the adjacent sections is the special feature of necrotic process in case of sharp leucosis, in consequence of which the unlimited ulcers of irregular outlines, covered with gray necrotic fur appear. Reactive changes around the ulcer are absent or are weakly expressed. The development of ulceronecrotic processes in the oral cavity is connected with sharp reduction in the resistibility of tissues caused by reduction in the phagocytic activity of leukocytes and immune properties of blood serum. Patients with acute leucosis complain about pain in the intact teeth and the jaws (together with pain in other bones), which is explained by the direct lesion of the bones in case of the leukemic process. Treatment is conducted in the hematology hospitals. The treatment of leukosis stomatitis is symptomatic. The extraction of teeth is contraindicated.

The chronic leucosis appear more rarely than acute, development is more fa- vorable; course is prolonged. Chronic myeloleukemia passes two stages: benign (lasts several years) and malignant (terminal), which lasts for 3 -6 months. The basic sign of chronic my- eloleukemia in the oral cavity are hemorrhagic manifestations, but with consider- ably smaller intensity, than in case of acute leucosis. Bleeding of gums appears not spontaneously, but only with the traumatization, the removal of teeth. In the period of excerbation the ulceronecrotic lesions of oral mucosa are observed. Chronic lymphoid leukosis is characterized by slow beginning and prolonged latent course. In the initial stage of disease an increase in groups of lymph nodes is observed. At the developed stage the generalized increase of lymph nodes is observed, the pallor of the skin and the mucous membranes, leukemic infiltrations of gums. the tongue, and hyperplasia of interdental papillae appear. Sometimes the growth of gingival edge reaches the level of the joining of teeth. Treatment is conducted in the hematology departments. Local treatment consists in care of the oral cavity, full-fledged sanitation, symptomatic therapy.

Agranulocytosis is a syndrome, which is characterized by the significant de- crease in the number or absence of neutrophilic granulocytes in the peripheral blood. Ulceronecrotic process in the lips, the gums, the tongue, mucous membrane of cheeks and other sections develop. Ulceronecrotic process can spread into the gullet. The absence of the inflammatory reaction of tissues around the centre of necrosis is im- portant for diagnostics. Frequently necrotic process is combined with candidiasis. Treatment is conducted in the hematology departments. Local

treatment is symptomatic; it includes the antiseptic treatment of oral cavity, anesthetization, removal of necrotic tissues, and prescription of the preparations, which stimulate regeneration.

## TOPIC 71.

## DIFFERENTIAL DIAGNOSTICS OF MANIFESTATIONS OF BLOOD DISEASES AND ORGANS OF BLOOD FORMATION ON A MUCOUS MEMBRANE OF AN ORAL CAVITY. ANEMIA. DENTIST'S TACTICS.

Manifestations on oral mucosa with the diseases of the blood and hematopoietic organs Iron-deficiency (hypochromic) anemia (chlorosis).

Patients with early chlorosis complain about the disturbance of gustatory senses, sense of smell, reduction in appetite, nausea. The oral mucosa is without the noticeable disturbances, except for color change it becomes pale. All symptoms are more expressed with late chlorosis. Patients complain about painful sensations in the tongue and oral mucosa when eating sour or spicy food, dryness in the oral cavity, paresthesia (burn- ing, tingling, prickling and bursting the tongue open), and the presence of angular heilitis.

The clinical picture of hypochromic anemia is multiple lesions of carious teeth, their increased abrasion, and loss of the natural luster of enamel. Mucosa membrane of lips, cheeks and gums is pale, edematic. The tongue is edematic, papillae are atrophied, espe- cially in its front part. It becomes clear red and smooth, as if polished, deep folds appear. Sometimes hemorrhages and cracks in the angles of the mouth are observed.

Treatment. The symptomatic therapy of manifestations on mucous membrane is prescribed. Hypoplastic anemia appears under the action of the exogenous physical (ir- radiation) and chemical factors, and drugs as well as endogenous aplasia of bone marrow. On the background of particularly pale oral mucosa different heavy hem- orrhages appear. Interdental papillae are edematic, cyanotic, sometimes bleeding, deep parodontal pockets are revealed. On oral mucosa, besides petechiae, also ero- sions, ulcers, necrotic sections are observed. Treatment. Dentist conducts the symptomatic treatment of the manifestations of hypoplastic anemia.

B12 - folate deficient anemia (malignant anemia, pernicious anemia, Addison – Birmera disease)

is characterized by the disturbance of erythropoiesis. The clinical picture of disease consists of the triad: the dysfunction of the gastrointestinal tract, hemopoietic and nervous systems. One of the early symptoms of the disease is the pallor of the skin and oral mucosa with a yellowish tint. Sometimes on oral mucosa petechial hemorrhages are observed. The most characteristic lesion of oral mucosa in case of malignant anemia is hunter's glossitis. The back of the tongue in this case takes the form of the smooth, bright, polished surface as a result of the atrophy of mushroom-shaped filamentary papillae, thinning of epithelium and atrophy of muscles. Painful, sharply limited strips and spots of clear red color of inflammatory nature appear at the back and the tip of the tongue. Treatment is carried out in the hematology clinics. Locally symptomatic treat- ments, sanitation of oral cavity arecarried out.

## TOPIC 72.

## DIFFERENTIAL DIAGNOSTICS OF MANIFESTATIONS OF BLOOD DISEASES AND ORGANS OF HEMATOPOIESIS ON THE ORAL MUCOSA. THROMBOCYTOPENIC PURPURA. DENTIST'S TACTICS

Erythremiya (polycythemia, the Vaquez' disease) Erythremiya appears at the age of 40-60 years, predominantly in men. Disease begins unnoticeably, and develops slowly. Increased fatigue, bleeding of gums, hemorrhages from the nose are noted. Characteristic symptom for patients is dark-cherry polycythemia of oral mucosa. Lips, the tongue, buccal mucosa are clear red because of the increased content of reduced hemoglobin in the capillaries. Mucous membrane in the region of alveolar branches is friable, with the cyanotic tint; when pressing bleeds easily, interdental papillae are hyperemized, and of dark- cherry colour. Expressive color boundary - cyanosis of soft palate and pale color of hard palate (Cooperman's symptom) is characteristic. The itching of the skin and paresthesia of oral mucosa due to the increased filling of vessels and the irritation of the interoceptors of capillaries by the blood is possible. Treatment is conducted by hematologist, making periodic bloodlettings and us- ing the cytostatic therapy with radioactive phosphorus or mielosan.

Thrombocytopenic purpura (Verlgof disease) The basic clinical symptom of disease is hemorrhages from the skin and oral mu- cosa, as well as nose and gums, that appear spontaneously or under the effect of the insignificant injury. In general in case of thrombocytopenic purpura the oral mucosa is pale, edematic, atrophied; the thinning of epithelium, erosions or ulcers form. Treatment is conducted in the hematology departments. Changes in the oral mucosa at hypo- and avitaminoses

## TOPIC 73.

## DIFFERENTIAL DIAGNOSIS OF ALLERGY-RELATED COMPLICATIONS OF DRUG THERAPY. DENTIST'S TACTICS. EMERGENCY ASSISTANCE.

A drug allergy is an allergic reaction to a medication. With an allergic reaction, your immune system, which fights infection and disease, reacts to the drug. This reaction can cause symptoms such as rash, fever, and trouble breathing.

True drug allergy is not common. Less than 5 to 10 percent of negative drug reactions are caused by genuine drug allergy. The rest are side effects of the drug. All the same, it's important to know if you have a drug allergy and what to do about it.

Different drugs have different effects on people. That said, certain drugs do tend to cause more allergic reactions than others. These include:

-antibiotics such as penicillin and sulfa antibiotics such as sulfamethoxazole-trimethoprim

-aspirin

-nonsteroidal anti-inflammatory medications, such as ibuprofen

-anticonvulsants such as carbamazepine and lamotrigine

-drugs used in monoclonal antibody therapy such as trastuzumab and ibritumomab tiuxetan

-chemotherapy drugs such as paclitaxel, docetaxel, and procarbazine

Why do drug allergies happen?

Your immune system helps protect you from disease. It's designed to fight foreign invaders such as viruses, bacteria, parasites, and other dangerous substances. With a drug allergy, your immune system mistakes a drug that enters your body for one of these invaders. In response to what it thinks is a threat, your immune system begins to make antibodies. These are special proteins that are programmed to attack the invader. In this case, they attack the drug.

This immune response leads to increased inflammation, which can cause symptoms such as rash, fever, or trouble breathing. The immune response might happen the first time you take the drug, or it may not be until after you've taken it many times with no problem.

## TOPIC 74.

## DIFFERENTIAL DIAGNOSIS OF COMPLICATIONS OF DRUG THERAPY ASSOCIATED WITH DYSBIOSIS. DENTIST'S TACTICS. EMERGENCY ASSISTANCE.

Dysbiosis is a condition in which the gut bacteria become imbalanced, leading to a wide range of digestive disturbances including bloating, diarrhea, constipation, and stomach cramps, among others. This condition has been linked to various illnesses including irritable bowel syndrome (IBS), inflammatory bowel disease, and gastritis, to name a few.

What is Dysbiosis?

Dysbiosis is a condition when the gut bacteria become imbalanced. As a result, a wide range of digestive disturbance symptoms occurs, including diarrhea, cramping, constipation, bloating, and indigestion. When there is a disparity in the gut’s normal flora, caused by too few beneficial bacteria and an overgrowth of bad bacteria, it can cause dysbiosis.

There are at least 400 species of bacteria found in the gut microbiome. They are essential for overall health as they aid in digestion, fight off pathogenic microorganisms, and synthesize vitamins. The normal flora of the body can be found in various areas and they’re essential for overall health and wellness. When these bacteria become imbalanced, and the bad ones override the beneficial bacteria, or grow uncontrollably, it can cause illness.

What Causes Ddysbiosis?

There are many factors that can lead to the condition, including the excessive or wrong use of antibiotics, excessive alcohol consumption, increased intake of sugar or protein, frequent use of antacids, exposure to pesticides, and chronic stress, to name a few. Also, poor dental hygiene and anxiety can also lead to dysbiosis.

In some cases, studies have linked dysbiosis to being born via C-section and being formula fed among newborns.

Signs and Symptoms of Dysbiosis

The main signs and symptoms of dysbiosis are digestive disturbances. People with the condition may experience frequent gas or bloating. This means that they feel bloated on most days of the week. Also, they suffer from abdominal cramping, diarrhea, and constipation, with mucus in the stool. They may have a combination of diarrhea and constipation, food sensitivities, food intolerances, and chronic bad breath.

## TOPIC 75.

## DIFFERENTIAL DIAGNOSIS OF COMPLICATIONS OF DRUG THERAPY ASSOCIATED WITH INTOXICATION. DENTIST'S TACTICS. EMERGENCY ASSISTANCE.

Intoxication is the term used to describe any change in perception, mood, thinking processes and motor skills that results from the effect of a drug(s) on our central nervous system.

intoxication is a transient condition of altered consciousness and behavior associated with recent use of a substance. It is often maladaptive and impairing, but reversible. If the symptoms are severe, the term "substance intoxication delirium" may be used ...

Symptoms of a drug overdose (including alcohol poisoning) may include:

-nausea and vomiting.

-severe stomach pain abdominal cramps.

-diarrhoea.

-chest pain.

-dizziness.

-loss of balance.

-loss of co-ordination.

-being unresponsive, but awake

However, there are some things they can do to feel more alert and appear soberer.

Coffee. Caffeine may help a person feel alert, but it does not break down alcohol in the body. ...

Cold showers. Cold showers do nothing to lower BAC levels. ...

Eating and drinking. ...

Sleep. ...

Exercise. ...

Carbon or charcoal capsules

## TOPIC 76.

## DIFFERENTIAL DIAGNOSIS OF MANIFESTATIONS ON THE MUCOUS MEMBRANE OF THE ORAL CAVITY OF DERMATOSES WITH AN AUTOIMMUNE COMPONENT.

Pemphigus vulgaris / Pemphigus vulgaris /, in which the oral mucosa lesions may be the only manifestation of the disease. Elements of defeat more often localized on the soft palate, retromolar in the cheek area , on the floor of the mouth, lips, gums and throat. Disease begins suddenly from formation of acantholytic, non-inflammatory bubbles that appear on the apparently unchanged or slightly hyperaemic oral mucosa diameter from 2-3 to 4 cm or more. First, bubbles filled with a clear liquid that becomes lemon hue after 1-2 days, and then becomes muddy. First, bubbles strained, but eventually become flabby. Bubbles become pear-shaped and quickly burst, with excerpts layers of the epithelium at the periphery under the weight of exudate. The doctor is rarely possible watch blisters, especially on the oral mucosa. Due to bursting of the bubble, stagnant red, clean, or covered with a light fibrinous plaque , painful erosion occurs. Pemphigus vegetans / Pemphigus vegetans /. It is characterized by the presence of necrotic epithelium oval or polygonal shape, weakly bound with the underlying tissue, and therefore resembles necrotic plaque, which can be easily removed with a cotton swab. Bottom erosion first looks uneven because of the rapid formation of growths (vegetations) that easily bleed. Profuse discharge from the surface of the lesions are easily decomposed, which is accompanied by an unusually unpleasant odor. New bubbles are formed on the periphery. The symptom of Nikolsky is positive. Pemphigus foliaceous / Pemphigus foliaceus / starts suddenly on a picture of normal general condition. Elements of defeat such as flaccid blisters or scabs are localized mainly on the face, scalp, and may be on the whole body. Bubbles merged and form erosive surface resembling a skin burn. Loose layers of the epidermis remain in their place, and under them new bubbles are formed. The symptom of Nikolsky is positive. General condition is significantly impaired. Body temperature increased. Oral mucosa is rarely affected. Seborrheic (erythematous) Pemphigus - Pemphigus erythematosus is very rare, resembles both lupus erythematosus and seborrheic dermatitis. Disease begins with skin lesions such as erythematous cells on a picture of oily seborrhea, with layering of dark or yellow crusts. On the oral mucosa lesions is the form of typical blisters localized on the mucous membrane of the cheeks, palate, pharynx, tongue, lips. Due to trauma of oral mucosa bubbles do not saved for a long time, after which the membranes white or greasy plaque are formed; during their rejection erosive surface strips.Sometimes the whole surface of the cheek or the palate looks like a continuous erosion, or coated by fibrous films. The rash may be localized on an unchanged mucosa.Diagnostics of pemphigus is based on history, clinical, cytological and histological examination.Typical clinical picture of pemphigus is a rash monomorphism, thin-walled bubbles, unresponsiveness of surrounding tissues, no remission, positive symptom of Nikolsky, a significant violation of the general condition of the patient, languid epithelialization of erosions,the presence of Ttsank cells. Differential diagnosis of all forms of true pemphigus performed with polymorphic exudative erythema , chronic recurrent aphthous stomatitis , lupus erythematosus , herpes stomatitis , dermatitis herpetiformis Duhring , herpes zoster , candidiasis , drug-induced , papular syphilis, pemfigoid form of planus , as well as self – non-acantolitic pemphigus and between different forms of true pemphigus .Treatment of pemphigus held by GCS: prednisolone, dexamethasone, triamptsinolon. Large doses of ascorbic acid up to 3 g per day, pentotenat calcium - 50 mg of calcium chloride to 2-3 grams per day, Pananginum, potassium orotate are also prescribed. Local treatment direct on preventing secondary infection, reduce pain and

epithelialization. For cleaning of plaque erosions used proteolytic enzymes, and then treated by Vinylinum , usnitatom sodium emulsion with sangviritrin corticosteroid ointments.Lichen planus ( CPL ) / Lichen ruber planus /. Elements of defeat at CPL are papules found on the skin, the red

border of the lips, oral mucosa.On the red border rash represents individual papules small size, polygonal shape. These papules are grouped and interconnected keratinised bridges. Papules keratinise, resulting in a few rise above the surrounding red border. Keratinised papule's top gives it a whitish or gray-white color.Papules often localized on the oral mucosa at the CPL . Here papules are military, grouped in the lines, strips, mesh, lace plexus, often forming a dendritic pattern , or a fern pattern or frost on the glass. Keratinize papule's tops give them a white milky color.Typical localization of elements of defeat is distal cheek or retromolar area where papules arranged in a fern leaf or Wickham's grid, specifically limited and may be palpated.Rarely papules are found on the tongue / dorsal and lateral surface / merging into plaque. Patients with KPL does not have any complaints certain time. Pain appears in violation of the integrity of the epithelium - erosive, ulcer and bullous /pemfigoid / form. Differential diagnosis of lichen planus is performed with hyperplastic candidiasis, papular secondary syphilis , drug allergies, pemphigus , toxic allergic action of drugs / krizanol - preparations of gold / ; leukoplakia , lupus erythematosus.Treatment includes causal, pathogenic and symptomatic therapy and divided into two groups: I - the overall treatment and II - local therapy. For topical treatment of patients prescribed rinse solution of hydrogen sulfide, citral, mouthparts bath of decoction of flax. Keratosis areas treated by retinol, sea buckthorn oil, karatolin, tocopherol or tsigerol before eating. Apply a 5 % solution of salicylic acid as a keratolytic agent.In the complex treatment of erosive form, together with conventional therapy of planus prescribe delagil and prednisolone, as well as a multivitamin, solkoseril, plasmol or splenin . For the treatment of erosive surface used filmy compositions from metatsil, mefenaminat natrium based on medical adhesive and collagen film. Ointment or jelly "Solkoseril ", as well as irradiation helium-neon laser promotes epithelialization of erosions. In order to speed up the repair process in the oral mucosa vacuum - phonophoresis with 0.1% levamisole is carried out.In the case of protracted erosive - ulcerous form of CPL when ulcer NOT heal up for 3 - weeks after therapy, diathermocoagulation or blizkofokusnaya radiotherapy are used to remove it. For a total treatment using sedation drugs (bromides, Bekhterev mixture, electrophoresis with bromine to collar of Shcherbakov, diathermy of cervical sympathetic ganglia) are used. To affect nonspecific reactivity prescribe pentoxy or prodigiozan and preparations of arsenic / sodium arsenate, 1 ml under the skin, 25-30 injections/, asian pills , multivitamins .When exudative - congestive and erosive ulcerous form used metronidazole (0, 25 x3 times a day 10 days).Systemic lupus erythematosus (a disease of Liebman - Sax) - connective tissue disease, which manifests itself by defeating a number of organs and systems. The disease is caused by disorders of immunological processes in the body, in which the antibodies produced by the body, damage DNA of healthy cells. Lupus is more common in young women. The etiological cause is chronic viral infection (RNA viruses similar to measles). Leading role in the pathogenesis of the disease takes a violation of humoral and cellular immunity, formation of circulating autoantibodies, particularly antinuclear antibodies to the integral kernel and its components, forming circulating immune complexes , especially DNA antibodies to DNA - complement. They deposit to the basement membranes of tissues of different organs, causing them to be damaged with the development of inflammation, which is associated with impaired cellular immunity, provided by T - lymphocytes, which are killed by the virus, lymphocytosis toxic antibodies, etc.. Family genetic predisposition may be. Insolation, pregnancy, childbirth, abortion, early menstruation, infection, reaction to the introduction of drugs, vaccines, and others provoke outbreak of disease.

## TOPIC 77.

## DIFFERENTIAL DIAGNOSIS OF RED SQUAMOUS LICHEN. CLINIC, DIAGNOSIS, TACTICS OF THE DENTIST. DIFFERENTIAL DIAGNOSIS OF PEMPHIGUS. CLINIC, DIAGNOSIS, TACTICS OF THE DENTIST.

Lichen planus ( CPL ) / Lichen ruber planus /. Elements of defeat at CPL are papules found on the skin, the red border of the lips, oral mucosa.On the red border rash represents individual papules small size, polygonal shape. These papules are grouped and interconnected keratinised bridges. Papules keratinise, resulting in a few rise above the surrounding red border. Keratinised papule's top gives it a whitish or gray-white color.Papules often localized on the oral mucosa at the CPL . Here papules are military, grouped in the lines, strips, mesh, lace plexus, often forming a dendritic pattern , or a fern pattern or frost on the glass. Keratinize papule's tops give them a white milky color.Typical localization of elements of defeat is distal cheek or retromolar area where papules arranged in a fern leaf or Wickham's grid, specifically limited and may be palpated.Rarely papules are found on the tongue / dorsal and lateral surface / merging into plaque. Patients with KPL does not have any complaints certain time. Pain appears in violation of the integrity of the epithelium - erosive, ulcer and bullous /pemfigoid / form. Differential diagnosis of lichen planus is performed with hyperplastic candidiasis, papular secondary syphilis , drug allergies, pemphigus , toxic allergic action of drugs / krizanol - preparations of gold / ; leukoplakia , lupus erythematosus.Treatment includes causal, pathogenic and symptomatic therapy and divided into two groups: I - the overall treatment and II - local therapy. For topical treatment of patients prescribed rinse solution of hydrogen sulfide, citral, mouthparts bath of decoction of flax. Keratosis areas treated by retinol, sea buckthorn oil, karatolin, tocopherol or tsigerol before eating. Apply a 5 % solution of salicylic acid as a keratolytic agent.In the complex treatment of erosive form, together with conventional therapy of planus prescribe delagil and prednisolone, as well as a multivitamin, solkoseril, plasmol or splenin . For the treatment of erosive surface used filmy compositions from metatsil, mefenaminat natrium based on medical adhesive and collagen film. Ointment or jelly "Solkoseril ", as well as irradiation helium-neon laser promotes epithelialization of erosions. In order to speed up the repair process in the oral mucosa vacuum - phonophoresis with 0.1% levamisole is carried out.In the case of protracted erosive - ulcerous form of CPL when ulcer NOT heal up for 3 - weeks after therapy, diathermocoagulation or blizkofokusnaya radiotherapy are used to remove it.Pemphigus vegetans / Pemphigus vegetans /. It is characterized by the presence of necrotic epithelium oval or polygonal shape, weakly bound with the underlying tissue, and therefore resembles necrotic plaque, which can be easily removed with a cotton swab. Bottom erosion first looks uneven because of the rapid formation of growths (vegetations) that easily bleed. Profuse discharge from the surface of the lesions are easily decomposed, which is accompanied by an unusually unpleasant odor. New bubbles are formed on the periphery. The symptom of Nikolsky is positive. Pemphigus foliaceous / Pemphigus foliaceus / starts suddenly on a picture of normal general condition. Elements of defeat such as flaccid blisters or scabs are localized mainly on the face, scalp, and may be on the whole body. Bubbles merged and form erosive surface resembling a skin burn. Loose layers of the epidermis remain in their place, and under them new bubbles are formed. The symptom of Nikolsky is positive. General condition is significantly impaired. Body temperature increased. Oral mucosa is rarely affected. Seborrheic (erythematous) Pemphigus - Pemphigus erythematosus is very rare, resembles both lupus erythematosus and seborrheic dermatitis. Disease begins with skin lesions such as erythematous cells on a picture of oily seborrhea, with layering of dark or yellow crusts. On the oral mucosa lesions is the form of typical blisters localized on the mucous membrane of the cheeks, palate, pharynx, tongue, lips. Due to trauma of oral mucosa bubbles do not saved for a long time, after which the membranes white or greasy plaque are formed; during their rejection erosive surface strips.Sometimes the whole surface of the cheek or the palate looks like a continuous erosion, or

coated by fibrous films. The rash may be localized on an unchanged mucosa.Diagnostics of pemphigus is based on history, clinical, cytological and histological examination.Typical clinical picture of pemphigus is a rash monomorphism, thin-walled bubbles, unresponsiveness of surrounding tissues, no remission, positive symptom of Nikolsky, a significant violation of the general condition of the patient, languid epithelialization of erosions, the presence of Ttsank cells. Differential diagnosis of all forms of true pemphigus performed with polymorphic exudative erythema , chronic recurrent aphthous stomatitis , lupus erythematosus , herpes stomatitis , dermatitis herpetiformis Duhring , herpes zoster , candidiasis , drug-induced , papular syphilis, pemfigoid form of planus , as well as self – non-acantolitic pemphigus and between different forms of true pemphigus .Treatment of pemphigus held by GCS: prednisolone, dexamethasone, triamptsinolon. Large doses of ascorbic acid up to 3 g per day, pentotenat calcium - 50 mg of calcium chloride to 2-3 grams per day, Pananginum, potassium orotate are also prescribed. Local treatment direct on preventing secondary infection, reduce pain and epithelialization. For cleaning of plaque erosions used proteolytic enzymes, and then treated by Vinylinum , usnitatom sodium emulsion with sangviritrin corticosteroid ointments.

## TOPIC 78.

## DIFFERENTIAL DIAGNOSIS OF LIP DISEASES. MODERN METHODS OF TREATMENT AND PREVENTION.

Cheilitis is a medical condition characterized by inflammation of the lips. According to its onset and course, cheilitis can be either acute or chronic.

| | |
|---|---|
| clinical manifestations , treatment and prognosis of meteorological cheilitis | Mashkill l Eyson AL was first described as an independent disease . Ill mostly men aged 20-75, the okiv , working in adverse weather conditions. Red border , preferably lower lip , not clearly hyperemic, infiltrated, dry, covered with small scales. CO lips without changes. The course is chronic and depends on the time of year. . **Histologically:** diffuse irregular epithelial hyperplasia, sometimes with a slight keratinization. |
| | **Treatment:** weaken the effect of meteofactors , locally anti-inflammatory ointments , for general treatment - vitamins of group B2 , B6, B12 and oil solution of vitamin A 10 drops 3 times a day for a month.<br>**Prognosis** : favorable when the patient is less in the open air. In the long run, malignancy is possible. |
| clinical manifestations , treatment and prognosis of actinic cheilitis | This is a chronic disease caused by the increased sensitivity of the red lip border to sunlight, resulting in a delayed type allergic reaction. First written by S. Aeres in 1923 . More often men 20-60 years get sick.<br>**Dry form:** hot lips, red border of lower lip bright red, covered with dry, small, silvery-white scales. Once removed, they reappear. The red lip of the lips is rough, easily injured. In the long run it is possible to develop cracks, erosion.<br>**The exudative form is** characterized by acute inflammatory phenomena: pain, red border edging, hyperemia, lip swelling, blisters, erosion, painful cracks, crusts. A characteristic feature of actinic cheilitis is the absence of damage to the corners of the mouth.<br>**Treatment:** protection of lips from sunlight , antihistamines, imudon, applications of anti-inflammatory ointments , keratoplasty.<br>**Prediction :** 1% of cases emerged and is malignancy of the lips . Active observation and timely treatment makes the prognosis favorable. |

| | |
|---|---|
| clinical manifestations , treatment and prognosis of contact allergic cheilitis | Contact allergic second cheilitis first described Miller - Taussing in 1925. The vast majority of patients are women aged 20-60 years. The disease occurs under the action of chemicals that are components of lipstick, components of toothpastes, powders, plastics of dentures. The disease can also be professional.<br>Characteristic is hotness, itching, mild pain, hyperemia, swelling, blistering, erosion, wetting. In milder cases, there is slight hyperemia, dryness, tightness of the lips, flaking, slight edema of the red lip border , cracks on the border with the skin. Provocative test (effect of elimination) - 5-7 days after the disappearance of acute events repeated use of a single factor , cause relapse.<br>**Treatment:** abolishing the use of the factors that led to the appearance of cheilitis, the application of anti-inflammatory ointments. General treatment: antihistamines.<br>**The prognosis is** favorable if allergen contact is not allowed again. |
| clinical manifestations , treatment and prognosis of of chronic lip fracture | On the lower lip poodyn exhibit at mo , linear defects of varying depth, length 1.5 cm, sometimes covered with bloody crust. Cracks can pass to the mucous membrane of the lip. Cracking without treatment is long-lasting (up to 3-8 years), with alternation of remissions and relapses. T rischyny hlybshayut formed painful infiltration, the edges are sealed, cell hyperkeratosis<br>. **Histologically:** chronic inflammation that is accompanied |
| | with the regenerative, hyperplastic, and sometimes metaplastic epithelial growth .<br>**Treatment:** to conservative and surgical . Conservative - AE Vit, a complex of B vitamins internally. Topical use of keratoplastics, antimicrobials, anti-inflammatory ointments in combination with antibiotics, adhesive dressings , blockages 0.25 - 0.5 ml of 1% novocaine, helium-neon laser. Surgical - cryodestruction.<br>**The prognosis is** favorable, but with long-term development of precancerous diseases (leukoplakia, skin horn) is possible. |

Meteorological cheilitis ( cheilitis meteorologic a) was first described as an independent disease by ALMashkillayson. Diagnosis of cheilitis is complex and requires a differential diagnosis with contact allergic, atopic, exfoliative (dry form), actinic, lupus erythematosus and red spotted lichen. The prognosis is favorable when the patient is less in the open air or changes his place of work, the signs of the disease diminish and even disappear. In the long run it becomes a background for the development of precancerous diseases, malignancy is possible.

Treatment :

- it is not necessary to reduce the influence of factors that caused the disease (hygienic lipstick, creams "Lux", "Delight", "Spermacetic")

- for general treatment, prescribe vitamins B 2 , B 6 , B 12 , nicotinic acid and oil vitamin A 10 drops 3 times a day for a month

- and corticosteroid-based ointments (senator and others)

- For the correction of the immune system - Imudone, as well as IgA in tablets under the tongue for resorption 7- 8 times a day.

Actinic cheilitis (cheilitis actinica) is a chronic condition that is caused by the increased sensitivity of the red lip border to sunlight. First described by S. Ayres in 1923. Differential diagnosis is required with exfoliative cheilitis, contact allergic and atopic cheilitis.

Treatment :

- protection of lips from sunlight (creams "Ray", "Shield", aerosol "Fencortozol")

- chingamine 0.25 1 time a day, starting in the spring, gradually increasing the dose to 2-3 tablets a day

- sIgA under the tongue before resorption (8 times a day )

- application of ointment fluoro for Horta ketaloha, sinalara, lokortena and others.

Actinic cheilitis is the background for the occurrence of precancerous malignancies. Hence the need for active surveillance, timely treatment, which makes the prognosis favorable.

Contact allergic cheilitis ( cheilitis venenata , s cheilitis allergica contactilis ). First described in 1925 by Miller - Taussing . Diagnosis is based on a clear identification of allergic history. The setting of skin tests is complicated because the skin reacts differently than the red border. What matters is the provocative test (elimination effect): 5-7 days after the disappearance of acute effects, repeated use of lipstick causes recurrence. Differential diagnosis should be made with actinic, meteorological, exfoliative, eczematous and atopic cheilitis.

Treatment . The use of the factors that led to cheilitis, the administration of corticosteroid ointment applications (senator, canine, etc.) 5-6 times a day is abolished. With a significant inflammatory reaction, prescribe intra-antihistamines (fincarol, clarithin, etc.). The prognosis is favorable if allergen contact is not allowed again.

Glandular cheilitis ( cheilitis glandularis ) is an inflammatory disease of the displaced into the transitional zone (Klein zone) and sometimes into the red border, small glands. He first introduced the term into Volkman's medical literature in 1870. Diagnosis is not difficult due to the peculiar clinic. Differential diagnosis , depending on the form, should be made with secondary forms of glundular cheilitis occurring on the background of red spotted lichen, leukoplakia, tuberculous and red lupus, with polycystosis, with lymphoedematous and granulomatous cheilitis. The prognosis for glondular cheilitis is favorable. But it should be remembered that there may be a precancerous disease of red lip border.

Treatment

is surgical and conservative.

In inflammatory processes:

- corticosteroid ointments with antibiotics (Si setting H, lococorten H, hyoxyzone)

- in purulent inflammation, antibiotics per os and topically (5%, 10% sintomitsinovoy emulsion fulevyl, Levosin 3% tetratsyk linova eritromitsynova or ointment)

- correction of the immune system: IIU forth on, sIgA.

Chronic cleft lip (rhagas labia chronica) is a limited inflammation that results in aslit-like linear defect in the epithelium and its own plate.Diagnosis of chronic cleft lip is not complicated. The prognosis is favorable, butover the long-term it becomes the background for the development of precancerous diseases (leukoplakia, skin horn). Treatment of chronic cracks is conservative and surgical. For conservative therapy use vitamin preparations (aevit, a complex of B vitamins), which are administered per os. In lesions use keratoplasty (retinol, aevit, rosehip oil, sea buckthorn, methyluracil ointment), antimicrobials (10% syntomycin emulsion, levovinisol, levomekol, etc.), corticosteroid ointments in combination with dibiotic, antibiotic, corticosterol, antibiotics dressings based on biological glue, as well as the blockade of 0.25-0.5 ml of 1% novocaine in the base of the crack on the oral side - 1-2 blockades with an interval of 5-7 days, heliumneon laser. In the conditions of appearance at cracks of scar atrophy of its edges or hyperkeratosis surgical removal of the lesion is shown. Exfoliative cheilitis ( cheilitis exfoliativa ). It was first described by Stelwagou (1900) under the name "transient lip desquamation". Mikulicz and K ü mmel (1912) named it exfoliative cheilitis. It is an xudative form. Diagnosis is usually not difficult: typical localization of the lesion, no erosion, characteristic appearance of the crusts. Differential diagnostics should be performed with eczematous, actinic, abrasive precancerous Manganotti cheilitis, erosive-ulcerative form of red spotted lichen and lupus erythematosus, vulgar blistering, multiform exudative erythema. Dry form Differential diagnosis should be made with meteorological, atopic, actinic (dry form) and atrophic fungal cheilitis.

Treatment. General treatment (should be performed with an endocrinologist, psychoneurologist):

- sedatives (infusion of valerian, hermit, peony);

- tranquilizers (Sebazon, Phenozepam, Eleniums, Mezapam);

- in severe depressive states - antidepressant drugs (amitriptyline, azafen);

- antihistamines (fencorol, clarithin);

- vitamins C and group B in therapeutic doses;

- Immune and sIgA in tablets up to 8 times a day are prescribed for the immune

system to be corrected, kept in the mouth until absorbed.

Locally in the lesion area use :

- corticosteroid ointments (triacort, fluorocort, senator, canalologist);

- in the presence of microbial flora - hormonal ointments with antibiotics

(dermozolone, dexocort, corticomycetin, fencortisol).

In case of ineffectiveness of conservative therapy, Bucca beams are prescribed: 1 Gy - 1 time per week up to 2-3 Gy at intervals of 7-10 days. Course dose from 10 to 12-20 Gy. Eczema cheilitis (cheilitis exzematosa) - a chronic recurrent allergic disease of red border and lip skin. There are isolated, eczematous lesions of the lips, but more often it is a symptom of skin eczema. Diagnosis is facilitated by the fact that there is a classic skin lesion. In other cases, the diagnosis of eczematous cheilitis is based on the presence of microvesicles, point-like serous wells, evolutionary polymorphism of lesions. Differential diagnosis should be made with contact allergic, meteorological actinic, atopic, exfoliative cheilitis.

Treatment of eczematous cheilitis complex:

- sedative therapy;

- antihistamines;

- low doses of corticosteroids and antibiotics (with microbial eczema);

- immunocorrector immunoconduct, sIgA;

- Gintosuggestive therapy, electrosleep.

Local:

- corticosteroid ointments, aerosols (lococorten, flucinar, fluorocort,

celestoderm, triderm).

Atopic cheilitis (cheilitis atopicalis) is a symptom of atopic dermatitis or neurodermatitis. Diagnosed more often in children 7-17 years. Diagnosis does not cause much difficulty. Attention should be paid to lesions of the skin of the neck, extremities (lesions of the hamstrings and elbows). Help to correctly diagnose changes in peripheral blood: lymphocytosis, eosinophilia, decrease in the number of E-lymphocytes, T-suppressors, increase in B-lymphocytes, hyperproduction of IgE. Differential diagnostics . Atopic cheilitis should be differentiated with exfoliative, contact allergic, meteorological, actinic, eczematous, mycotic, and streptococcal angular cheilitis. The outlook is favorable.

Treatment of atopic cheilitis is complex .

General :

- antihistamines;

- vitamins (B 2 , B 6 );

- tranquilizers (Elenium, Seduxen, Tazepam);

- hypo sensitizing histoglobulin therapy during the acute phase of the disease

- for the correction of the immune system: imudon, sIgA;

- for severe disease - a short course of corticosteroid therapy (prednisone,

dexamethasone);

- diet (excluding salty, spicy, spicy foods, alcohol, reducing carbohydrates);

- spa treatment in dry and warm climates.

Local Interventions:

- corticosteroid ointments (fluorocort, senator, flucinar, betnovate, locolortene);

- for lichenization - application of 10-20% ichthyol ointment or 10% naphthylanov

liniment;

- physical procedures: magnetotherapy, infrared rays, laser therapy.

## TOPIC 79.

## DIFFERENTIAL DIAGNOSIS OF DISEASES OF THE TONGUE. MODERN METHODS OF TREATMENT AND PREVENTION. PHYSICAL METHODS OF DIAGNOSIS AND TREATMENT OF DISEASES OF THE TONGUE AND LIPS.

Geographic tongue.

The cause of geographical language can be a number of diseases:

-Diseases of the gastrointestinal tract;

-Disorders of B vitamins (B1, B2, B6, B12);

-Diseases of the stomach and duodenum (gastritis, gastroduodenitis, peptic ulcer);

-Malabsorption syndrome (violation of food absorption in the small intestine);

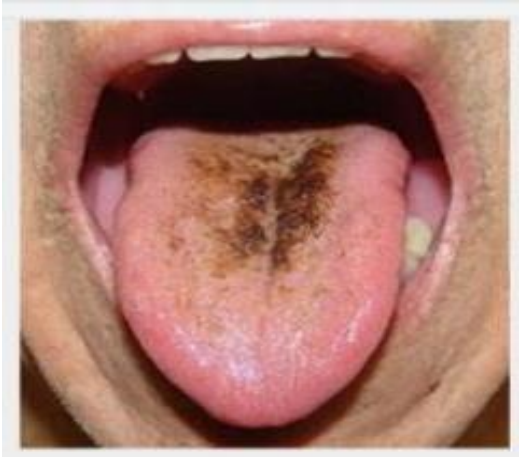
-Liver disease;

-Diseases of the pancreas (pancreatitis, diabetes, tumors;

-Some pathologies of the endocrine system (diseases of the thyroid gland, pancreas, adrenal glands);

Symptoms

The process usually begins with the appearance on the tongue of a gray area of turbidity with a diameter of several millimeters, which eventually swells, and in the center of it peel off filamentous papillae, exposing the red area of rounded shape. The peeling zone increases rapidly, while maintaining a level rounded shape. The intensity of desquamation (exfoliation) decreases. Desquamation spots can be of different sizes and shapes. Sometimes they have the shape of rings and semi-rings. in the zone of desquamation mushroom-shaped papillae in the form of bright red points are well visible



Causes of black tongue The disease, as mentioned earlier, occurs most often in middle-aged and older men. In this regard, we can identify specific favorable factors that can provoke the following disease:

Smoking abuse.

Tobacco smoking has a particularly strong effect.

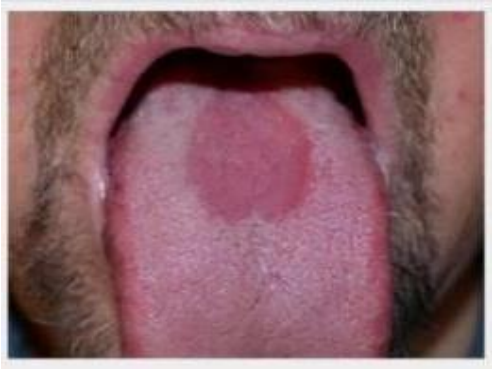
Abuse of coffee and strong tea.

Other reasons: Violations of oral hygiene, poor care of teeth and tongue. The use of some drugs, namely - drugs containing bismuth, as well as taking antibiotics. Water imbalance, dehydration, hyposalivation - insufficient amount of saliva. Irradiation of the head.

Treatment of black tongue

The main thing in the treatment of this disease is to restore oral hygiene and carefully monitor cleanliness. Brush your teeth at least twice a day, preferably after each meal.

Also, the patient should increase the amount of fluid consumed and give up bad habits and drinking coffee and tea. If the development of black tongue is caused by fungi or bacteria, the doctor should prescribe appropriate courses of antibiotics or antifungal drugs.



Symptoms and signs

The external manifestations of rhomboid glossitis of the tongue depend on the form of the disease, but the general symptoms are characteristic of all types of glossitis.

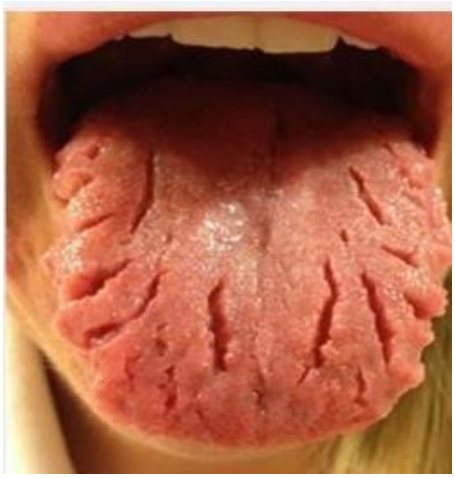
At the initial stage, the pathology manifests itself only in the form of lesions of the back of the tongue: the formation in the form of a diamond or oval of bluish-red hue has a length of not more than 2 centimeters and a width of not more than 5 centimeters.

The middle form is accompanied by a feeling of a foreign body in the mouth, swelling of the tongue, increased salivation. Diction disorders are possible.

As the disease progresses, the symptoms worsen: ulcers appear, taste sensations disappear. The patient has difficulty talking, eating and even drinking, at the slightest impact there is a sharp piercing pain.

Treatment of the smooth form does not involve surgery, only the appointment of the necessary drugs. At bumpy and papillomatous type the acting fabrics are cut off. To begin with, the patient undergoes complete rehabilitation of the oral cavity - removes plaque, tartar, caries and other pathologies to eliminate pathogenic bacteria. Then prescribe antifungal and antiseptic drugs, a course of vitamins, healing ointments. If the reason was a disorder of the stomach, a special diet is recommended.

Folded (scrotal) tongue - a congenital anomaly of the shape and size of the tongue, which is expressed in the presence of deep grooves (folds) running in different directions.



Symptoms

Folding is often accompanied by a moderate increase in the whole language - macroglossia. The presence of numerous furrows on its surface is characteristic. The longitudinal fold is usually located strictly in the middle, originating from the tip of the tongue and often reaching the level of the location of the grooved papillae, from it depart transverse folds (in the form of leaf veins).

Melkersson–Rosenthal syndrome (also termed "Miescher-MelkerssonRosenthal syndrome"),[1] is a rare neurological disorder characterized by recurring facial paralysis, swelling of the face and lips (usually the upper lip), and the development of folds and furrows in the tongue.[2]:799 Onset is in childhood or early adolescence. After recurrent attacks (ranging from days to years in between), swelling may persist and increase, eventually becoming permanent. The lip may become hard, cracked, and fissured with a reddish-brown discoloration. The cause of Melkersson–Rosenthal syndrome is unknown, but there may be a genetic predisposition. It has been noted to be especially prevalent among certain ethnic groups in Bolivia. It can be symptomatic of Crohn's disease or sarcoidosis.Treatment

Treatment is symptomatic and may include nonsteroidal antiinflammatory drugs (NSAIDs) and corticosteroids to reduce swelling, antibiotics and immunosuppressants. Surgery may be indicated to relieve pressure on the facial nerves and reduce swelling, but its efficacy is uncertain. Massage and electrical stimulation may also be prescribed.

Melkersson–Rosenthal syndrome may recur intermittently after its first appearance. It can become a chronic disorder. Follow-up care should exclude the development of Crohn's disease or sarcoidosis.

There are obvious causes of red color change of the tongue, such as something you ate (strawberries or red-colored foods). Some acidic foods can cause temporary redness and discomfort. However, a raspberry-colored tongue can be a sign of an underlying medical condition. Some red color changes on the tongue ("strawberry tongue") could be related to a vitamin deficiency, Kawasaki disease, or a strep infection (scarlet fever).

Erythroplakia is a red area or lesion that cannot be rubbed off on the tongue (except for the color, it is similar to leukoplakia). A lesion with a combined white and red appearance is called erythroleukoplakia. These lesions are all considered to have premalignant potential. Erythroplakia and erythroleukoplakia have an increased risk of premalignancy compared to leukoplakia. In

addition to appearance, there is concern if the lesion or sore does not go away or grows in size. A biopsy is recommended by an oral surgeon or an ENT specialist to rule out oral cancer

## TOPIC 80.

## DIFFERENTIAL DIAGNOSIS OF PRECANCEROUS CONDITIONS OF THE MUCOUS MEMBRANE OF THE ORAL CAVITY AND RED LIP BORDER.

Malignant pre-cancer diseases

Classification

• Bowen's disease (Keira Erytroplaziya);

• Premalignant warty;

• Limited precancerous hyperkeratosis of

red lipborder;

• Abrasive prekantseroz cheilitis

Manhanotti;

Classification

II. Facultative precancerous high

probability of malignancy:

• Lekoplakiya;

• Papillomatosis;

X-ray stomatitis;

• Erosive and ulcerative form of lupus

erythematosus;

• Keratoakantoma;

• Cutaneous horn.

Classification

III Facultative pre-cancers with less

possibility of malignance:

• Flat leucoplaky.

• Oral mucosa chronic ulcers.

• Red flattened lichen and red lupus erosive-ulcerous and hyperkeratic forms.

• Lips chronic fissures.

• Postradial cheilitis and stomatitis.

• Meteorological and actynial cheilites.

Classification of pretumor processes of oral mucosa

With high frequency of malignization (obligate): 1) morbus Bowen.

With little frequency of malignization (facultative): 1) verrucuos and erosive leukokeratosis; 2) polypapilloma; 3) erosive ulcerous and hyperkeratotic forms of erythema centrifugum and lichen acuminatus; 4) postradial stomatitis.

Classification of pretumor processes of red border

With high frequency of malignization (obligate):

1) verrucous precarcinoma; 2) limited precancerous hyperkeratosis; 3) abrasiva praecancerosa cheilitis Manganotti.

With little frequency of malignization (facultative): 1) leukokeratosis; 2) kera-toacanthoma; 3) cutaneous horn; 4) papilloma with comification; 5) erosive ulcerous and hyperkeratotic forms of erythema centrifugum and lichen acuminatus; 6) postradial cheilitis.

Leukoplakia

Leukoplakia generally refers to a firmly attached white patch on a mucous membrane which is associated with an increased risk of cancer. The edges of the lesion are typically abrupt and the lesion changes with time. Advanced forms may develop red patches. There are generally no other symptoms. It usually occurs within the mouth, although sometimes mucosa in other parts of the gastrointestinal tract, urinary tract, or genitals may be affected.

Leukoplakia is a descriptive term that should only be applied after other possible causes are ruled out. Tissue biopsy generally shows increased keratin build up with or without abnormal cells, but is not diagnostic. Other conditions that can appear similar include yeast infections, lichen planus, and keratosis due to repeated minor trauma. The lesions from a yeast infection can typically be rubbed off while those of leukoplakia cannot.

Treatment recommendations depend on features of the lesion. If abnormal cells are present or the lesion is small surgical removal is often recommended; otherwise close follow up at three to six month intervals may be sufficient. People are generally advised to stop smoking and limit the drinking of alcohol. In potentially half of cases leukoplakia will shrink with stopping smoking; however, if smoking is continued up to 66% of cases will become more white and thick. The percentage of people affected is estimated at 1–3%. Leukoplakia becomes more common with age, typically not occurring until after 30. Rates may be as high as 8% in men over the age of 70.

Definition

The word leukoplakia means "white patch", and is derived from the Greek words λευκός - "white" and πλάξ - "plate". Leukoplakia is a diagnosis of exclusion, meaning that which lesions are included depends upon what diagnoses are currently considered acceptable. Accepted definitions of leukoplakia have changed over time and are still controversial. It is possible that the definition will be further revised as new knowledge becomes available.

In 1984 an international symposium agreed upon the following definition: "a whitish patch or plaque, which cannot be characterized clinically or pathologically as any other disease, and is not associated with any physical or chemical agent except the use of tobacco." There were however problems and confusion in applying this definition. At a second international symposium held in 1994 it was argued that whilst tobacco was a likely causative factor in the development of leukoplakia, some white patches could be linked directly to the local effects of tobacco by virtue of their disappearance following smoking cessation, suggesting that this kind of white patch represents a reactive lesion to local tissue irritation rather than a lesion caused by carcinogens in cigarette smoke, and could be better termed to reflect this etiology, e.g. smokers' keratosis. The second

international symposium therefore revised the definition of leukoplakia to: "a predominantly white lesion of the oral mucosa that cannot be characterized as any other definable lesion."

In the mouth, the current definition of oral leukoplakia adopted by the World Health Organization is "white plaques of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer". However, this definition is inconsistently applied in the medical literature, and some refer to any oral white patch as "leukoplakia".

The term has been incorrectly used to describe white patches of any cause (rather than specifically referring to idiopathic white patches) and also to refer only to white patches which have a risk of cancerous changes. It has been suggested that leukoplakia is an unhelpful term since there is so much inconsistency surrounding its use, and some clinicians now avoid using it at all.

Classification

- Leukoplakia in the lower labial sulcus
- Leukoplakia of the soft palate
- Exophytic leukoplakia on the buccal mucosa
- Leukoplakia on the side of tongue

Leukoplakia could be classified as mucosal disease, and also as a premalignant condition. Although the white color in leukoplakia is a result of hyperkeratosis (or acanthosis), similarly appearing white lesions that are caused by reactive keratosis (smoker's keratosis or frictional keratosis e.g. morsicatio buccarum) are not considered to be leukoplakias. Leukoplakia could also be considered according to the affected site, e.g. oral leukoplakia, leukoplakia of the urinary tract, including bladder leukoplakia or leukoplakia of the penis, vulvae, cervix or vagina. Leukoplakia may also occur in the larynx, possibly in association with gastro-esophageal reflux disease. Oropharyngeal leukoplakia is linked to the development of esophageal squamous cell carcinoma, and sometimes this is associated with tylosis, which is thickening of the skin on the palms and soles of the feet (see: Leukoplakia with tylosis and esophageal carcinoma). Dyskeratosis congenital may be associated with leukoplakia of the oral mucosa and of the anal mucosa.

Mouth

Within the mouth, leukoplakia is sometimes further classified according to the site involved, e.g. leukoplakia buccalis (leukoplakia of the buccal mucosa) or leukoplakia lingualis (leukoplakia of the lingual mucosa). There are two main clinical variants of oral leukoplakia, namely homogenous leukoplakia and non-homogenous (heterogenous) leukoplakia, which are described below. The word leukoplakia is also included within the nomenclature of other oral conditions which present as white patches, however these are specific diagnoses which are generally considered separate from leukoplakia, with the notable exception of proliferative verrucous leukoplakia, which is a recognized sub-type of leukoplakia.

Homogenous leukoplakia

Homogenous leukoplakia (also termed "thick leukoplakia") is usually well defined white patch of uniform, flat appearance and texture, although there may be superficial irregularities. Homogenous leukoplakia is usually slightly elevated compared to surrounding mucosa, and often has a fissured, wrinkled or corrugated surface texture, with the texture generally consistent throughout the whole lesion. This term has no implications on the size of the lesion, which may be localized or extensive. When homogenous leukoplakia is palpated, it may feel leathery, dry, or like cracked mud.

Non-homogenous leukoplakia

Non-homogenous leukoplakia is a lesion of non-uniform appearance. The color may be predominantly white or a mixed white and red. The surface texture is irregular compared to homogenous leukoplakia, and may be flat (papular), nodular or exophytic. "Verrucous leukoplakia" (or "verruciform leukoplakia") is a descriptive term used for thick, white, papillary lesions. Verrucous leukoplakias are usually heavily keratinized and are often seen in elderly people. Some verrucous leukoplakias may have an exophytic growth pattern, and some may slowly invade surrounding mucosa, when the term proliferative verrucous leukoplakia may be used. Non-homogeneous leukoplakias have a greater risk of cancerous changes than homogeneous leukoplakias.

Proliferative verrucous leukoplakia

Proliferative verrucous leukoplakia (PVL) is a recognized high risk subtype of non-homogenous leukoplakia. It is uncommon, and usually involves the buccal mucosa and the gingiva (the gums). This condition is characterized by (usually) extensive, papillary or verrucoid keratotic plaques that tends to slowly enlarge into adjacent mucosal sites. An established PVL lesion is usually thick and exophytic (prominent), but initially it may be flat. Smoking does not seem to be as strongly related as it is to leukoplakia generally, and another dissimilarity is the preponderance for women over 50. There is a very high risk of dysplasia and transformation to OSCC or to verrucous carcinoma.

Erythroleukoplakia ("speckled leukoplakia"), left commissure. Biopsy showed mild epithelial dysplasia and candida infection. Antifungal medication may turn this type of lesion into a homogenous leukoplakia (i.e. the red areas would disappear)

Erythroleukoplakia (also termed speckled leukoplakia, erythroleukoplasia or leukoerythroplasia) is a non-homogenous lesion of mixed white (keratotic) and red (atrophic) color. Erythroplakia (erythroplasia) is an entirely red patch that cannot be attributed to any other cause. Erythroleukoplakia can therefore be considered a variant of either leukoplakia or erythroplakia since its appearance is midway between. Erythroleukoplakia frequently occurs on the buccal mucosa in the commissural area (just inside the cheek at the corners of the mouth) as a mixed lesion of white nodular patches on an erythematous background, although any part of the mouth may be affected. Erythroleukoplakia and erythroplakia have a higher risk of cancerous changes than homogeneous leukoplakia.

Signs and symptoms

Most cases of leukoplakia cause no symptoms, but infrequently there may be discomfort or pain. The exact appearance of the lesion is variable. Leukoplakia may be white, whitish yellow or grey. The size can range from a small area to much larger lesions. The most common sites affected are the buccal mucosa, the labial mucosa and the alveolar mucosa, although any mucosal surface in the mouth may be involved. The clinical appearance, including the surface texture and color, may be homogenous or non-homogenous. Some signs are generally associated with a higher risk of cancerous changes.

Causes

The exact underlying cause of leukoplakia is largely unknown, but it is likely multifactorial, with the main factor being the use of tobacco. Tobacco use and other suggested causes are discussed below. The mechanism of the white appearance is thickening of the keratin layer, called hyperkeratosis. The abnormal keratin appears white when it becomes hydrated by saliva, and light

reflects off the surface evenly. This hides the normal pink-red color of mucosae (the result of underlying vasculature showing through the epithelium). A similar situation can be seen on areas of thick skin such as the soles of the feet or the fingers after prolonged immersion in water. Another possible mechanism is thickening of the stratum spinosum, called acanthosis.

Tobacco

Tobacco smoking or chewing is the most common causative factor, with more than 80% of persons with leukoplakia having a positive smoking history. Smokers are much more likely to suffer from leukoplakia than non-smokers. The size and number of leukoplakia lesions in an individual is also correlated with the level of smoking and how long the habit has lasted for. Other sources argue that there is no evidence for a direct causative link between smoking and oral leukoplakia. Cigarette smoking may produce a diffuse leukoplakia of the buccal mucosa, lips, tongue and rarely the floor of mouth. Reverse smoking, where the lit end of the cigarette is held in the mouth is also associated with mucosal changes. Tobacco chewing, e.g. betel leaf and areca nut, called paan, tends to produce a distinctive white patch in a buccal sulcus termed "tobacco pouch keratosis". In the majority of persons, cessation triggers shrinkage or disappearance of the lesion, usually within the first year after stopping.

Alcohol

Although the synergistic effect of alcohol with smoking in the development of oral cancer is beyond doubt, there is no clear evidence that alcohol is involved in the development of leukoplakia, but it does appear to have some influence. Excessive use of a high alcohol containing mouth wash (> 25%) may cause a grey plaque to form on the buccal mucosa, but these lesions are not considered true leukoplakia.

Ultraviolet radiation

Ultraviolet radiation is believed to be a factor in the development of some leukoplakia lesions of the lower lip, usually in association with actinic cheilitis.

Micro-organisms

Candida in its pathogenic hyphal form is occasionally seen in biopsies of idiopathic leukoplakia. It is debated whether candida infection is a primary cause of leukoplakia with or without dysplasia, or a superimposed (secondary) infection that occurs after the development of the lesion. It is known that Candida species thrive in altered tissues. Some leukoplakias with dysplasia reduce or disappear entirely following use of antifungal medication. Smoking, which as discussed above can lead to the development of leukoplakia, can also promote oral candidiasis. Candida in association with leukoplakia should not be confused with white patches which are primarily caused by candida infection, such as chronic hyperplastic candidiasis ("candidal leukoplakia").

The involvement of viruses in the formation of some oral white lesions is well established, e.g. Epstein-Barr virus in oral hairy leukoplakia (which is not a true leukoplakia). Human papilloma virus (HPV), especially HPV 16 and 18, is sometimes found in areas of leukoplakia, however since this virus can be coincidentally found on normal, healthy mucosal surfaces in the mouth, it is unknown if this virus is involved in the development of some leukoplakias. In vitro experimentation has demonstrated that HPV 16 is capable of inducing dysplastic changes in previously normal squamous epithelium.

Epithelial atrophy

Leukoplakia is more likely to develop in areas of epithelial atrophy. Conditions associated with mucosal atrophy include iron deficiency, some vitamin deficiencies, oral submucous fibrosis, syphilis and sideropenic dysphagia.

Trauma

Another very common cause of white patches in the mouth is frictional or irritational trauma leading to keratosis. Examples include nicotine stomatitis, which is keratosis in response to heat from tobacco smoking (rather than a response to the carcinogens in tobacco smoke). The risk of malignant transformation is similar to normal mucosa. Mechanical trauma, e.g. caused by a sharp edge on a denture, or a broken tooth, may cause white patches which appear very similar to leukoplakia. However, these white patches represent a normal hyperkeratotic reaction, similar to a callus on the skin, and will resolve when the cause is removed. Where there is a demonstrable cause such as mechanical or thermal trauma, the term idiopathic leukoplakia should not be used.

Differential diagnosis

There are many known conditions which present with a white lesion of the oral mucosa, but the majority of oral white patches have no known cause. These are termed leukoplakia once other likely possibilities have been ruled out. There are also few recognized subtypes of leukoplakia, described according to the clinical appearance of the lesion.

Almost all oral white patches are usually the result of keratosis. For this reason oral white patches are sometimes generally described as keratoses, although a minority of oral white lesions are not related to hyperkeratosis, e.g. epithelial necrosis and ulceration caused by a chemical burn. In keratosis, the thickened keratin layer absorbs water from saliva in the mouth and appears white in comparison with normal mucosa. Normal oral mucosa is a red-pink color due the underlying vasculature in the lamina propria showing through the thin layer of epithelium. Melanin produced in the oral mucosa also influences the color, with a darker appearance being created by higher levels of melanin in the tissues (associated with racial/physiologic pigmentation, or with disorders causing melanin overproduction such as Addison's disease). Other endogenous pigments can be overproduced to influence the color, e.g. bilirubin in hyperbilirubinemia or hemosiderin in hemochromatosis, or exogenous pigments such as heavy metals can be introduced into the mucosa, e.g. in an amalgam tattoo.

Leukoplakia cannot be rubbed off the mucosa, distinguishing it readily from white patches such as pseudomembranous candidiasis, where a white layer can be removed to reveal an erythematous, sometimes bleeding surface underneath. The white color associated with leukoedema disappears when the mucosa is stretched. A frictional keratosis will generally be adjacent to a sharp surface such as a broken tooth or rough area on a denture and will disappear when the causative factor is removed. Some have a suggested as general rule that any lesion that does not show signs of healing within 2 weeks should be biopsied. Morsicatio buccarum and linea alba are located at the level of the occlusal plane (the level at which the teeth meet). A chemical burn has a clear history of placing an aspirin tablet (or other caustic substance such as eugenol) against the mucosa in an attempt to relieve toothache. Developmental white patches usually are present from birth or become apparent earlier in life, whilst leukoplakia generally affects middle aged or elderly people. Other causes of white patches generally require pathologic examination of a biopsy specimen to distinguish with certainty from leukoplakia.

Management

A systematic review found that no treatments commonly used for leukoplakia have been shown to be effective in preventing malignant transformation. Some treatments may lead to healing

of leukoplakia, but do not prevent relapse of the lesion or malignant change. Regardless of the treatment used, a diagnosis of leukoplakia almost always leads to a recommendation that possible causative factors such as smoking and alcohol consumption be stopped, and also involves long term review of the lesion, to detect any malignant change early and thereby improve the prognosis significantly.

Predisposing factors and review

Beyond advising smoking cessation, many clinicians will employ watchful waiting rather than intervene. Recommended recall intervals vary. One suggested program is every 3 months initially, and if there is no change in the lesion, then annual recall thereafter. Some clinicians use clinical photographs of the lesion to help demonstrate any changes between visits. Watchful waiting does not rule out the possibility of repeated biopsies. If the lesion changes in appearance repeat biopsies are especially indicated. Since smoking and alcohol consumption also places individuals at higher risk of tumors occurring in the respiratory tract and pharynx, "red flag" symptoms (e.g. hemoptysis - coughing blood) often trigger medical investigation by other specialties.

Surgery

Surgical removal of the lesion is the first choice of treatment for many clinicians. However, the efficacy of this treatment modality cannot be assessed due to insufficient available evidence. This can be carried out by traditional surgical excision with a scalpel, with lasers, or with eletrocautery or cryotherapy. Often if biopsy demonstrates moderate or severe dysplasia then the decision to excise them is taken more readily. Sometimes white patches are too large to remove completely and instead they are monitored closely. Even if the lesion is completely removed, long term review is still usually indicated since leukoplakia can recur, especially if predisposing factors such as smoking are not stopped.

Medications

Many different topical and systemic medications have been studied, including anti-inflammatories, antimycotics (target Candida species), carotenoids (precursors to vitamin A, e.g. beta carotene), retinoids (drugs similar to vitamin A), and cytotoxics, but none have evidence that they prevent malignant transformation in an area of leukoplakia. Vitamins C and E have also been studied with regards a therapy for leukoplakia. Some of this research is carried out based upon the hypothesis that antioxidant nutrients, vitamins and cell growth suppressor proteins are antagonistic to oncogenesis. High doses of retinoids may cause toxic effects. Other treatments that have been studied include photodynamic therapy.

Cornu cutaneum

Cornu cutaneum is limited hyperplasia of the epithelium with strong hyperkeratosis that resembles horn taking into account its appearance and solidity. It occurs on vermilion border, more often on lower lip, and is characteristic for people over 60, it is nontender. Its color is grey or grey-brown, diameter is up to 1 cm, length is up to 1 cm. Cutaneous horn is a long lasting disease (for years). Emergence of inflammation and induration around horn base, intensification of comification are the main signs of its malignization. The diagnosis is confirmed after removal of lesion and its histological examination. Surgical treatment is removal of cutaneous horn in the limits of healthy cells.

Keratoacanthoma

Keratoacanthoma is epidermal benign tumor that develops quickly and regresses spontaneously. The disease occurs on vermilion border, rarely on tongue. Keratoacanthoma appears

as a grey-red solid papule with choanoid deepening in the center filled with horn mass that can be easily removed. The tumor grows rapidly and in a month it reaches its maximum dimensions (2.5 x 1 cm).

Keratoacanthoma is nontender, movable and do not infiltrate surrounding tissues. In 6-8 months the tumor either regresses spontaneously and disappears leaving a scar or becomes malignant one and causes cancer. Keratoacanthoma must be distinguished from verrucous carcinoma and precancer. Cancer has firmer texture, solid base, after removal of horn masses bleeding emerges. Surgical treatment of keratoacanthoma is ligatory.

Morbus Bowen

Bowen was the first to describe this disease in 1912. From the very beginning it appears to be cancer in situ.

Clinical presentation. The affected area is as a rule single hyperemic bright red spot, smooth or with velutinate surface due to small papillary projections. The central area resembles leukokeratosis with pit-and-mount surface or lichen acuminatus with comification focuses on hyperemic background. Due to atrophy of' mucosa, the focus falls in slightly compare to surrounding areas, slightly bleeding erosions can appear on it. The affected area is from 1-2 mm to 5-6 cm, its contours are not smooth but rather distinct. Induration in its basis is not determined. If lesion is localized on the tongue, lingual papillae at affected area disappear. Regional lymph nodes are not palpable as a rule. Subjective sensations are minor but in cases of erosions tenderness can be present. Clinical presentation of Morbus Bowen on oral mucosa is not always apparent. The disease can manifest only by the small area of hyperemia or resemble leukokeratosis without evident inflammation. The disease may be present for indefinite time, in some cases invasive growth advances and traumatization accelerates this process, in other cases the disease remains at stage of cancer in situ. The diagnosis must be confined by histological study. Pathomorpbologically morbus Bowen is characterized by signs of intraepithelial epidermoid cancer: polymorphism of spicular layer cells up to atypia, increase of mitosis number, its impairment, presence of giant cells, multinuclear cells, acanthosis, and in several cases hyperkeratosis and parakerathosis. The basal membrane and basal layer are preserved. There is small infiltration with lymphocytes and plasmocytes at the upper part of stroma.

Differential diagnostics includes leukoplakia, lichen acuminatus, chronic traumatic affection.

Cheilitis abrasiva praecancrosa Manganotti

This type of precancerous lesions was identified and described by Manganotti in 1933. It occurs predominantly in men over 50 years. Traumas, insolation, herpes may cause the emergence of this disease.

Clinical presentation. Unlike low-grade limited or expanded chronic catarrhal inflammation of the lower lip one or rarely several red anabrosis with flat surface are seen. They can be sometimes covered with tight blood-tinged or serous crust, which is hard to remove. Anabrosis uncovered with scab tends to bleed. Induration at the base of the lesion is absent. Anabrosis can be hardly treated with ointments and applications of topical medications. Existing for a long time they can epithelize but then they appear again at the same or other areas. Histologically the deficiency of epithelium is observed as well as inflammatory infiltration in underlying connective tissue. Epithelium on the anabrosis edges is in the state of acanthosis or is atrophic. Epithelial bundles deviate deep into stroma. In some areas acanthocytes demonstrate different rate of discomplement and atypia. Cytological examination can reveal the signs of dyskariosis of epithelial cells,

inflammatory elements but more often only inflammation. The process lasts from 1-2 months up to many years. If not treated it undergo malignization, which manifests clinically by induration of the anabrosis base and surrounded areas, emergence of papillary projections on the surface of anabrosis, its bleeding and comification around the anabrosis. The diagnosis is confirmed by revelation of atypical cells in scrapes from the diseased area or by results of histological examination. The differential diagnostics must be performed with anabrosis forms of leukoplakia, lichen acuminatus, erythema centrifugum, vesicular fever, herpes iris, actinic cheilitis, herpetic anabrosis.

Treatment. It is necessary to remove properly all local irritators, and then oral cavity sanation must be executed including adequate prosthesis, smoking and consumption of irritating food must be forbidden, removal of insolation is also recommended. It is necessary to reveal and to treat accompanying diseases of other organs and systems. Vitamin A is prescribed intraoraly (solution of retinol acetate in oil 3.44% or solution retinol palmitate in oil 5.5 %) 10 drops 2-3 times per day, as well as other polyvitamins. Applications with oil solution of vitamin A are prescribed topically, in case of baseline inflammation — ointments with corticosteroids and antibiotics. Nonsurgical therapy must not be carried out for more than 1 month. The best results are achieved by surgical removal of focus in the limits of healthy tissues. Only in case of Manganotti cheilitis the nonoperative treatment is permitted. The treatment of all types of obligate precancer is surgical procedure — full dissection of the diseased area in the limits of healthy tissues followed by immediate histological examination. Dissected tissue is surveyed by means of preparation of serial sections. Operation must be preceded by oral cavity sanation and removal of irritators. If operative treatment is impossible, radiation therapy is required.

## TOPIC 81.

## MODERN METHODS OF TREATMENT AND PREVENTION OF PRECANSEROUS LESSIONS.

Treatment

• Removal of traumatic factor.

• Vitamin A and vitamin B complex.

• Surgical.

• Cryodestruction.

Bowen disease – 1-2 times per year.

• Abrasive cheilitis Manganotti – 3 times per

year.

• Leucoplakia verrucous and erosive –

4 times per year.

• Lichen planus – 4 times per year.

• Lupus – 4 times per year.

Prevention of precancerous diseases

sanitary-hygienic – cancerogens discovery and action canceling:

• rational physical training,

• properfeeding especially as for vitamins,

• canceling alcohol taking and smoking,

• not to use hot and spicy food,

• refusal from other bad habits,

• preventing excessive insolation,

• hygienic care for oral cavity and lips red margin;

Prevention of precancerous diseases clinical:

• preventive examinations in the risk groups

• preventing and treatment concomitant diseases – somatic (herpetic injuries,

meteorological, actynial, glandular cheilites et al.);

• oral cavity sanation – to pay especial attention to traumatizing factors canceling;

• not to use cauterizing substance;

• dyspanserization

Prevention of precancerous diseases

• 1. Take steps to strengthen the body (rational mode of work, rest. Meals without alcohol abuse.

• 2. Avoid actions traumatic factors.

• 3. Early treatment of diseases (lupus erythematosus, lichen red clap, herpes simplex virus, streptococcal and fungal lesions, somatic diseases).

• 4. Rational use of vitamins for preventive, restorative purposes (A, B, PP).

• 5. Rational skin red border of lips (use fat mazovyh applications, chapstick lip with a high protective filter

## TOPIC 82.

## LABORATORY METHODS OF DIAGNOSTICS OF THE BASIC DENTAL DISEASES. SEROLOGICAL DIAGNOSTICS IN THERAPEUTIC DENTISTRY. METHODS OF COLLECTING MATERIAL FOR CYTOLOGICAL EXAMINATION AND ITS ANALYSIS.

Cytology is the exam of a single cell type, as often found in fluid specimens. It's mainly used to diagnose or screen for cancer. It's also used to screen for fetal abnormalities, for pap smears, to diagnose infectious organisms, and in other screening and diagnostic areas.

The cells to be examined may be taken through the following methods:

Scraping or brushing the tissue surface, such as during a pap smear

Collecting body fluids, such for urine or respiratory phlegm

## CONCLUSIONS

By complementing the study at the Faculty of Dentistry with the analysis and study of the information array of this study guide, each student will be able to improve the study of the discipline "Therapeutic Dentistry", namely to be ready to work in a dental clinic, to study the anatomical and histological features of the structure of oral mucosa, to distinguish between dental instruments and their functions, to develop the ability to treat different lesions of the oral mucosa in compliance with the relevant principles, to distinguish and be able to prepare dental materials what can help to surely diagnose, treat and prevent lesions of oral mucosa.

LIST OF REFERENCES

**Main literature**

1. Stomatology: textbook: in 2 books. Book I / M.M. Rozhko, Z.B. Popovych, V.D. Kuroiedova et al.: edited by M.M. Rozhko. – Kyiv: AUS Medicine Publishing, 2020. – 792 p.: color edition.
2. Periodontal and Oral Mucosa Diseases: textbook. Vol. 2 / A.V. Borsenko, L.V. Lynovytska, O.F. Nesyn et al.; edited by A.V. Borysenko. - Kyiv: AUS Medicine Publishing, 2018. – 624 p.; color
edition.
3. Stomatology: textbook: in 2 books. Book 2 / M.M. Rozhko, 1.I. Kyrylenko, O. H. Denysenko et al.; edited by M. M. Rozhko. – Kyiv: AUS Medicine Publishing, 2018. – 960 p.; color edition.

## Additional literature

1. Esthetic Dentistry: A Clinical Approach to Techniques and Materials 3rd Edition by Kenneth W. Aschheim. Publisher: Mosby; 3rd edition, 2014. – 600 p.
2. Esthetic and Restorative Dentistry: Material Selection and Technique 3rd Edition by Douglas A Terry, Willi Geller. Publisher: Quintessence Publishing Co Inc.; 3rd edition, 2017. – 776 p.
3. Dental Composite Materials for Direct Restorations Softcover reprint of the original 1st ed. by Vesna Miletic. Publisher: Springer; 1st edition, 2018. – 327 p.
4. Posterior Direct Restorations 1st Edition by Salvatore Scolavino, Gaetano Paolone. Publisher: Quintessence Pub Co; 1st edition, 2021. – 264 p.
5. Smile Design Integrating Esthetics and Function Essentials of Esthetic Dentistry Volume Two by Jonathan B. Levine. Elsevier Health Sciences, 2015. – 240 p.
6. Cohen's Pathways of the Pulp 12th Edition by Louis H. Berman, Kenneth M. Hargreaves. Publisher: Elsevier; 12th edition, 2020. –992 p.
7. Newman and Carranza's Essentials of Clinical Periodontology, 1st Edition. Michael G. Newman & Irina F Dragan & Satheesh Elangovan & Archana K. Karan. 2022. – 352 p.
8. Newman and Carranza's Clinical Periodontology, 13th Edition. Michael G. Newman & Henry Takei & Perry R. Klokkevold & Fermin A. Carranza. 2019. – 944 p.