Danylo Halytsky Lviv National Medical University
Pediatric Dentistry Department

Methodological Recommendations for Pediatric Therapeutic Dentistry for the 5th year students the 10thterm

Lviv 2021

Composed by:

Skybchyk O.V.; assist.; Kostura V.L., assist., PhD., Fur M. B., assist., PhD.

Chief Editor: Kolesnichenko O.V., Assoc. Prof., PhD

Reviewed by: Chukhray N.L., Prof.,Doctor of medical science

Ripetska O.R., Assoc. Prof., PhD

Manyuk L.V., senior lecturer

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Protocol № 1 from « 31 » _____August_____2021

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Responsible for the issue

Vice-Rector for Academic Affairs, Professor M.R. Grzegotskyy

THEMATIC PLAN OF THE PRACTICAL LESSONS
Pediatric Therapeutic Dentistry
5th year, 10th terms№

	Theme of the lesson	Hrs.
1	Content, structureand requirementsof testlicense examination "Krok-2" of Dentistry.Rules of solving the test problems.	7
2	The main direction of caries prevention. General and local cariogenic factors. Endogenic and exogenic caries prevention. Means and methods, and its effectiveness.	7
3	Peculiarities of the clinical course, diagnosis and treatment of caries of primary and permanent dentition in children. Modern filling materials, their features and choice in practice in Pediatric Therapeutic Dentistry.	7
4	Clinical course, diagnosis, and differential diagnosis of pulpitis of primary and permanent teeth in children. The choice of the method of treatment depending on the stage of development.	7
5	Clinical course, diagnosis and differential diagnosis of periapical inflammation of primary and permanent teeth in children. Modern endodontic instruments, peculiarities of endodontic treatment in teeth with unformed root. Prognosis.	7
6	Periodontal diseases in children. Gingivitis, periodontitis, periodontal syndrome. Etiology, pathogenesis, clinical course, diagnosis, differential diagnosis.	7
7	Main principles of periodontal diseases treatment in children. The choice of the medical remedies.	7
8	Differential diagnosis of diseases of oral mucosa in children (viral, bacterial, traumatic, allergic origin and during somatic diseases).	7
9	Main principles of treatment of diseases of oral mucosa in children (viral, bacterial, traumatic, allergic origin and during somatic diseases).	7
10	Summary module control.	7
	Total	70

THEMATIC PLAN OF THE SELF-WORK
Pediatric Therapeutic Dentistry
4th year, 7th term

№	Topic	Acad. Hrs.
1	Preparing for the practical lessons (theoretical, practical skills): topics 1-9	9
2	1. Pharmacodynamics of the main remedies that are used during the treatment of diseases of the periodontal tissue and oral mucose membrane.	2
	2. Radiologic methods of diagnosis in pediatric dentistry.	2
	3. Physiotherapeutic methods of treatment in pediatric dentistry.	2
	4. Odontogenic sources of infection in pediatric dentistry.	2
3	Preparing for the summary module control	3
	Total	20

PRACTICAL CLASS № 1

Theme: Content, structureand requirementsof testlicense examination “Krok-2” of Dentistry.Rules of solving the test problems.

Objective: Familiarize students with conduction of the licensing examination Krok-2 "Dentistry".

Contents of the class

Licensing examinations are conducted in accordance with the Regulations on the system integrated licensing examination of specialists with higher education trends "Medicine" and "Pharmacy", approved by the Ministry of Health of Ukraine from 14.08.1998 №251, of the organization and procedure of state certification students, approved by the Ministry of Health of Ukraine from 31.01.2005 №53, the order of the Ministry of Health of Ukraine dated 15.01.1999 №7 «on measures to implement the order of the Ministry of Health of Ukraine dated 14.08.1998 №251« on approval of the system of integrated licensing examination of the specialists with higher education on directions "Medicine" and "Pharmacy" Procedure of integrated licensing examination, agreed with the Ministry of Health of Ukraine 29.04.2013.

Licensing examination is conducted in written form (blank) testing. Examination consists of 200 tests items that previously passed the professional examination. Tests were formed into the so-called examination booklet: "The collection of tests for licensing exam," which includes tests format: situational problems, questions and five answers marked Latin letters (A, B, C, D, E). Only one correct answer.The 1 minute to 1 a test (200 minutes in total). Students - citizens of foreign countries have additional time (40 minutes) for testing.

Comprehension control

1. What is an integrated examination "Krok-2"?
2. Features admission to the examination.
3. Registration of students for passing the licensing examination.
4. The rights and obligations for passing the license examination.
5. Guidelines for passing the license examination.

Test control

1.Parents of 3.5 years old child complain of frequent nosebleeds, bleeding under the skin and mucous membranes in their child. Objectively: on the pale skin and oral mucosa the multiple petechiae are present. There is spontaneous bleeding of gums. What additional tests should be undertaken for the diagnosis?

A. Complete blood test
B. Biochemical examination of blood
C. Immunological examination of blood
D. Blood test for sugar
E. Urine test for sug

2. Child of 14 years old appealed to the dentist for dental sanation. Objectively: the skin of the face is pale, the rim of the lips is dry. The lips are covered with flakes. There are cracks in the corners of the mouth. The mucous membrane of the mouth is pale, the tongue is hyperemic and smooth, filiform papillae are atrophied. There is swelling and cyanosis of the gingival margin in the frontal area. MDF = 10. Enamel of the teeth is without brilliance. Clarify the diagnosis:

A. Iron deficiency anemia
B. Hemophilia
C. Werlhof's disease
D. Acute leukemia
E. Vitamin B12 Deficiency Anemia

3. The patient complains of headache, muscle ache and joint pain, fatigue, lack of appetite for a month. There is a massive bleeding of gums during teeth brushing. Also the patient complains of gingival overgrowth, its burning and sore, pain during eating. Objectively: paleness of the skin and mucous membrane. There are teeth imprints on the mucous membrane of the cheeks. There are petechial hemorrhage on the tongue and palate existing bleeding. The gums are hyperemic, edematous and loose in the frontal teeth area. Teeth are intact. Clarify the diagnosis:

A. Acute leukemia
B. Chronic leukemia
C. Hypertrophic gingivitis
D. Hypovitaminosis C
E. Werlhof disease

4. The patient appealed to the dentist with complaints of frequent bleeding of the mucous membrane of the mouth and nose. The patient specifies that the same problems have been observed in his father. Objectively: multiple existing telangiectasia and anhiomatous formations on skin and mucous membrane of cheeks, lips and tongue are noted. Blood test is within normal limits. What is the most likely diagnosis?

A. Osler-Weber-Rendu disease
B. Werlhof's disease
C. Schoenlein-Henock disease
D. Anemia
E. Von Willebrand disease

5. 15 years old girl complains of deteriorating of the health condition (fatigue, dizziness, headache, loss of appetite, nausea) during the last year. From history of disease revealed long (5-6 days) massive menstruation. Objectively: paleness and dryness of the skin, brittleness of the hair. Intraoral examination: cracks in the corners of the mouth, mucous membrane is pale and dry, filiform papillae of the tongue are atrophied. Blood test: Hb - 80 g/l, color index - 0,75, anisocytosis, hypochromia of erythrocytes. Preliminary diagnosis:

A. Iron deficiency anemia
B. Vitamin B 12 deficiency anemia
C. Hypoplastic anemia
D. Chronic leukemia
E. Hypovitaminosis of vitamin C

6. The boy of 17 years old complains of frequent nosebleeds, gums bleeding, positive braid symptom. Werlhof's diseases was diagnosed previously. Which index of blood test will confirm the diagnosis?

A. The number of platelets
B. Number of reticulocytes
C. The number of leukocytes
D. The amount of hemoglobin
E. ESR

7. Patient of 15 years old complains of gums bleeding. From history: frequent nosebleeds, weakness. Objectively: paleness of the skin and oral mucosa. On the mucous membrane of the cheeks, tongue and soft palate there are numerous petechiae. Blood test: red blood cells - 3.1 million, hemoglobin - 94 g/l, color index - 0.9, clotting time - 9 min, ESR 18 mm/h. What is the most likely diagnosis?

A. Iron deficiency anemia
B. Leukemia
C. Vaquez Osler disease

D. Addison - Birmer anemia
E. Werlhof's disease

8. A girl of 16 years old complains of burning, soreness of the tongue tip, impaired taste sensation, dry mouth. She suffers from antacid gastritis. Objectively: mucous membrane is pale and yellowish color. Cracks in the corners of the mouth are noted. There are teeth imprints on the mucous membrane on the cheek and filiform papillae are absent on the tip of the tongue. The back of the tongue is red. Blood test: Hb -80 g/l, color index - 0.7. What is the most likely diagnosis?

A. Folic and vitamin B12 deficiency anemia
B. Acute leukemia
C. Iron deficiency anemia
D. Werlhof's disease
E. Von Willebrand 's disease

9. The 7 years old boy's parents complained of the spontaneous night-time bleeding from the gums and nose in their son. The presence of small and large hemorrhages of different colors (from red to blue-green-yellow) in the mouth and on the skin of the child are noted. Blood test: significant decreasing of platelets and the presence of giant platelets in peripheral blood is observed. Clarify the diagnosis:

A. Werlhof's disease
B. Acute leukemia
C. Chronic leukemia
D. Hemophilia
E. Iron deficiency anemia

10. For what disease the following symptoms are significant: small symmetrical hemorrhages on the feet, legs, hips, painful symmetric polyarthritis, abdominal pain, hemorrhagic rash in the mouth, violation of the permeability of vascular walls?

A. Hemorrhagic vasculitis
B. Hemophilia C
C. Osler-Weber-Rendu syndrome
D. Werlhof's disease
E. Hemophilia A

Recommended literature

1. Paediatric dentistry/ Richard Welbury, Monty Duggal – 3rd ed., 2005 Copyright.
2. L.A. Khomenko. Pediatric Therapeutic Dentistry.- K.:Book-plus, 2012

PRACTICAL CLASS № 2

Theme: The main direction of caries prevention. General and local cariogenic factors.Endogenic and exogenic caries prevention.Means and methods, and its effectiveness.

Objective: Learn with students local and general cariogenic factors, means and things for conduction of the endogenic and exogenic prevention of caries on teeth, indications and method of conduction of the fissure sealing.

Pre-study test questions

1. Structure of the enamel, its chemical composition.

2. Sequence and terms of maturation of enamel of the different part of the tooth.
3. General cariogenic factors as a cause of tooth decay.
4. Local cariogenic factors in oral cavity.
5. Factors which influence on the development and mineralization of the teeth before and after their eruption.
6. Saliva, its composition and properties.
7. What is disturbance of resistance of the hard dental tissue?
8. Role of the superficial deposits on the teeth in caries development.
9. What is cariogenic situation in oral cavity.
10. Role of Fluoride and other micro elements in formation of enamel.
11. Types of fissures of teeth.
12. What is antenatal caries prevention.
13. Medicamentous prevention of pregnant women.
14. Role of Fluoride and other micro elements in caries development.
15. Role Fluoride, Calcium and other micro elements in caries prevention.
16. What is optimal concentration of the Fluoride in drinking water.

Contents of the class

The primary goal of preventive dentistry is to maintain the oral structures in a state of optimal health for the longest period of time possible - using the simplest, most universally acceptable methods.

The major concern in preventive dentistry is to stop the plaque-caused destruction of teeth by dental caries and the loss of their support through inflammatory periodontal disease.

However, all forms of dental neglect are costly in terms of pain, ill health, financial burden, loss of man-hours, psychological damage, and waste of human resources. Preventive dentistry can help to reduce or eliminate these costs.

Basic cariogenic factors of the oral cavity are microflora of the oral cavity (especially when the hygienical state of the oral cavity is unsatisfactory), uncontrolled use of carbohydrates, change of composition of saliva, insufficient functional loading of the tooth system. To the general cariogenic factors belong: low maintenance of fluor in a drinking-water, somatic state of a child's health, unfavorable ecological ways of residence. Cariogenic factors can have different degrees of intensity, so however much terms of the oral cavity are permanent and they depend on composition of a drinking-water, meal, functional state of the tooth-jaw system, state of organs and systems of all organism.

A cariogenic situation of the oral cavity always appears at high concentration of free hydrions (H^+), the source of which are organic acids (mainly milk) that is appeared during fermentation of carbohydrates by the microorganisms of dental plaque. The accumulation of microorganisms on the tooth surface to contribute: diminishing of self-cleaning of teeth, crowding of teeth, bad filling, prosthetic and orthodontic apparatus, violations of physical and chemical properties of saliva.

Cariogenic action of microorganisms explains the beginning of dental plaque. A dental plaque adjoins closely to the tooth surface and disposes above pellicle. A dental plaque contains the same basic types of microorganisms in different correlation in most of people. Thus, streptococci (mutans, salivarius, mitis, sangvis) makes about 40% from the common amount of microorganisms of the plaque.

A number of microorganisms can produce enough acid to decalcify tooth structure, particularly aciduric streptococci, lactobacilli, diphtheroids, yeasts, staphylococci, and certain

strains of sarcinae. Streptococcus mutans has been implicated as one of the major and most virulent of the caries –producing organisms.

It is generally accepted that the dental caries process is controlled to a large extent by a natural protective mechanism inherent within the **saliva**. Many features of saliva have been investigated in during caries process. Considerable importance has been placed on the salivary pH, the acid-neutralizing power, and the calcium, fluoride, and phosphorus content. It has been long suggested that in addition to these features the rate of flow and the viscosity of saliva may influence on the development of caries. The normal salivary flows aids to the solution of food debris on which microorganisms thrive. In addition, the saliva manifests a variety of antibacterial and other antiinfectious features. All well-known characteristics of saliva seem somehow are relevant to the process of dental caries.

The mucous membrane of the oral cavity, saliva, include the multicomponent system of defense from the pathogenic factors of environment. The epithelium of mucous membrane shows itself a functional barrier to the microorganisms, and from its state, above all things, defense of oral cavity depends from infections. But, most value in the defense mechanism of the oral cavity is acquired by the specific and heterospecific factors of resistance. Mechanic cleaning of oral cavity takes place due to saliva flow, the use of hard meal. Saliva hinders to adhesion of microorganisms to the surface of mucous membrane of oral cavity, to hard tissues of teeth. The IgA of saliva can change metabolism of microorganisms, limit formation of colonies, reduce virulence of contagiums.

Methods of determination of cariogenic situation in the oral cavity:

1. Methods, which are based on the study of tooth environment (lactobacilli test, determination of pathogenic microflora of dental plaque, determination of pH, viscidity, buffer capacity, mineral components of saliva

2. Methods that study solubility of enamel (TER-test (Okushko, Kosareva, 1983)). Teaching methodology: put the drop of a 1N solution of HCl on cleaned from the plaque, dried up, isolated from saliva vestibular surface of upper central incisor in the distance 2mm from a cutting edge. In 5 seconds wash off the acid, dry out an enamel. Put 1 drop 1% methylene dark blue on a bitten-into enamel, take off by a cotton tampon. Estimate a test on a 10-point scale. Fluoride varnish inflicted on the area of demineralization.

3. The individual risk of the development of caries is determined on the basis of estimation: rapid formation of dental plaque, level of the use of cariogenic products, the amount of initial caries.

During practical work students examine children and find out a soft dental plaque and the main features of saliva. Students solve situational tasks with the teacher.

Teacher makes conclusion at the end of the lesson. Teacher points out on student's drawbacks and their mistakes. Teacher announces the results.

Depending on the ways of influence on the hard tissues of tooth, the prevention is divided into endogenic, that influence through an organism, and exogenic, that influence directly on hard tissues of tooth, and also distinguish medicinal and unmedicinal prevention. The endogenic unmedicinal prevention of teeth decay includes strengthening of the somatic health of a child, the treatment of chronic diseases of internals and systems, rational feed with the limited use of carbohydrates.

The medicinal endogenic prevention is based on the reception of the preparations of fluor, calcium, phosphorus, microelements, vitamins A,C,D, preparations which influence on the immunological reactivity.

The removal of cariogenic situation is the purpose of local prevention of the teeth decay, adjusting of uncompleted, after eruption of teeth mineralization, focus on initial caries. The term “topical fluoride prevention” refers to the use of systems containing relatively large concentrations of fluoride that are applied locally, or topically, to the erupted tooth surfaces in order to prevent the formations of dental caries. This term encompasses the use of fluoride rinses, dentifrices, pastes, gels, and solution that are applied in various manners.

The local prevention is divided into medicinal and unmedicinal. An unmedicinal local prevention includes intensive mastication of the hard products, rational hygiene of oral cavity, rinse of oral cavity by solutions of natrium hydrocarbonate, xylite, mineral water, tea, slow drinking of the milk, tea. A medicinal local prevention includes local application of preparations of Ca, P, F (application, rinses).

Comprehension control

1What are the signscariogenicsituationin the mouth.

2What is the purposeof endogenousandexogenouscaries prevention.

3The role offluorineand othertrace elementsin the preventionof dental caries.

4. Calcium supplements used for endogenic prevention of tooth decay.

5. Which medications of phosphorus are used for endogenic caries prevention.

6. Which medications of fluoride are used for endogenic caries prevention

7. Medicamentous and non medicamentous endogenic caries prevention.

8. Vitamins used for endogenic caries prevention.

9. Technic of teeth fluoride varnishing?

10. Name the gels containing fluoride.

11. Method of rinsing the mouth with a solution of sodium fluoride?

12. What is the sequence of applications of fluoride gels and solutions of sodium fluoride on surfaces of teeth.

13. Describe the methods of the local use of medications containing calcium and phosphorus.

14. Name medications of remineralizing actions that are used for exogenic prevention.

15. Describe the technique of using medications of remineralizing action.

16. Indications for fissure sealing.

17. Methods of fissure sealing, materials.

18. Mistakes and complications for fissure sealing.

Test control

1. A 6- year- old child visited a dentist for a preventive examination. Objectively: mouth cavity is healthy. The 16,26,36,46 teeth are intact, erupted 3 months ago. Which of the known preventive methods should be used?

A. Fissure and pit silant

B. Applications of 10% Calcium gluconatis

C. lonophoresis of 1% NaF

D. Preparation "Vitafluorine"

E. A coating of fluorine varnish

2. During an examination of a 9-year-old child on the cervical surface of the 12, 11, 21 and 22 teeth white spots are found which 2 weeks ago appeared. The spots are without brilliance, and can

be colored with methylene. The affected teeth do not react to cold irritants. What should the dentist's tactic be to the affected teeth?

A. Remineralization therapy
B. Filling of the carious cavity
C. Polishing of the damaged areas
D. Impregnation therapy
E. Regular medical check-ups

3. A 16-year-old patient complains of the feeling of soreness in the lower teeth during 2 weeks. On examination: in the cervical area of the 11 and 21 teeth there are white spots with unclear contours which stain intensively. What treatment of the 11 and 21 teeth should be used?

A. Remineralization therapy
B. Antiseptic treatment
C. Preparation and filling
D. Impregnation therapy with argentic nitrate
E. Polishing of damaged areas

4. An 18 -year- old girl complains of the presence of numerous brown spots on the upper and lower teeth. She was born in the region where the maintenance of fluorine in a drinking-water was 2, 2 mg/1 and lived there for 10 years. What can prevent the hard tissues pathology development?

A. Using inward preparations of calcium and phosphorus
B. Introduction to the ration of sea products
C. Using inward preparations of fluorine
D. Treatment of teeth
E. Careful hygienic care of oral cavity

5. The very large teeth are the sign coupled with a Y-chromosome. The mother's teeth are of normal size, and her son has very large teeth. The probability of having very large teeth makes at a father:

A. 100%
B. 50%
C. 12.5%
D. 75%
E. 25%

6. The parents of a 6- year-old child visited a dentist for a preventive examination. Oral cavity is healthy. Recently, according to parents, the 36 and 46 teeth were erupted. What preventive methods should be used in the first 1.5-2 years after eruption of the indicated teeth?

A. Fissure and pit sealant
B. Covering of teeth with fluorine varnish
C. Impregnation therapy
D. Applications of Remodent
E. Rinsing of NaF

7. A mother of a 6.5 -year-old child visited a dentist for the check-up of fissure sealants on the 16, 26, 36 and 46 teeth which were made 6 months ago. The sealants were saved only in 36, 46 teeth. What will be the doctor's tactic?

A. Fissure sealing again
B. lonophoresis of 1 % NaF
C. To cover teeth with fluorine varnish
D. Application of Remodent solution
E. Preventive filling

8. During the prophylactic examination of a 13.5- year-old child in the area of the lower and upper frontal teeth a stagnant hyperemia and insignificant swelling of gingival margin was found. There is a periodic bleeding of gums during toothbrushing, DMF=2. What toothpastes are recommended for the individual hygiene of oral cavity in this case?

A. Toothpastes, which contain the extracts of medical plants
B. Toothpastes, which contain sodium fluoride
C. Toothpastes, which contain aminofluoride
D. Toothpastes, which contain calcium
E. Toothpastes, which contain salt additions

9. During the prophylactic examination of a 6-year-old child on the cusps of the 36 and 46 teeth the white spots with the clear margin are found. The teeth have been recently erupted. The enamel is transparent in these areas; during probing-smooth. What additional test is necessary to clarify the diagnosis?

A. Staining with methylene blue
B. X-ray
C. Staining with iodine solutions
D. Visiography
E. EOD

10. What type of fissure sealing do you know?

A. Invasive, noninvasive
B. Invasive
C. Noninvasive
D. Preventive filling
E. Invasive, Noninvasive, Preventive filling

Recommended literature

1. Paediatric dentistry/ Richard Welbury, Monty Duggal – 3rd ed., 2005 Copyright.
2. L.A. Khomenko. Pediatric Therapeutic Dentistry.- K.:Book-plus, 2012

PRACTICAL CLASS № 3

Theme: Peculiarities of the clinical course, diagnosis and treatment of caries of primary and permanent dentition in children. Modern filling materials, their features and choice in practice in Pediatric Therapeutic Dentistry.

Objective: Learn with students the peculiarities of the clinical course of caries of teeth in children of different age, methods of general treatment of caries, classification, content, properties, indications for using and methods of implementation of the modern filling materials for restoration of carious defects in the pediatric therapeutic dentistry.

Pre-study test questions

1. Formation and mineralization of teeth in the antenatal period, factors which influence onthese processes.
2. Stages of the development of the roots and term of the secondary mineralization of the primary and temporary teeth.
3. Stages of the preparation of the carious cavity.
4. Instruments for the filling of the cavity.
5. Classifications of the filling materials.
6. Instruments for filling processing.

Contents of the class

Dental caries, also known as **tooth decay** or a **cavity**, is an infection, bacterial in origin, that causes demineralization and destruction of the hard tissues of the teeth (enamel, dentin and cementum).

Treatment of caries in deciduous teeth can be performed without filling (non-operational treatment methods) or by the carious cavity preparation and filling, which is used in case of superficial, moderate and deep caries.

There are the following nonoperative methods of caries treatment in deciduous teeth:

- remineralization therapy;
- impregnation treatment method (silver impregnation)

Remineralization therapy is done with the use of medications containing ions of calcium, phosphates and fluoride. Taking into account the age of children and their behavior peculiarities in the treatment process, it is advisable to perform short time imanipulations - covering the affected enamel areas by fluoride varnishes, gels;deep fluoridation of enamel.

Impregnation treatment method (silver impregnation).Indications :

- acute and chronic superficial caries in primary teeth at all development stages;
- circular caries in primary teeth;
- caries of smooth-surface in primary teeth;
- approximal caries in the frontal group of primary teeth at stage of root stabilization and resorption.

Treatment methods. A 4%-solution of silver nitrate followed by 4%-solution hydroquinone for precipitation of unsolvable silver salts on the demineralized hard tissue surface, are used.

For the impregnation treatment of caries, the doctor removes dental plaque from the enamel surface of the affected deciduous teeth with an excavator , applies 3% solution of hydrogen dioxide and dries the surface; applies silver nitrate solution (with a cotton pellet) on the carious surface; applies a reducing agent for 1 —2 minutes. The manipulation is repeated 3—5 times. Silver impregnation of carious cavities in deciduous teeth is performed 3—4 times daily or every other

day. Impregnation courses are repeated every 3—4 months. A stable black coloring of affected tissues in deciduous teeth is a criterion of effective impregnation.

Treatment of caries in deciduous teeth by preparation and filling is used in case of superficial, moderate and deep caries in deciduous teeth. It consists of preparation and formation of a carious cavity, and its filling with a suitable sealing material.

Carious cavity preparation in deciduous teeth is done with:

– high-speed and mechanic handpieces and burs of various sized and configurations;

– chemical-mechanical method (covering of carious dentine with a special gel and manual removal of softened dentine with special instruments or an excavator);

– manual preparation with excavators of various sizes (ART-method).

Opening of carious cavity is removal of overhanging enamel edges aimed to create an easy access for the carious cavity examination followed by preparation. To perform this manipulation the doctor uses a turbine hand piece, fissure or round diamond bur.

Necretomy is started by a sharp excavator, matched to the carious cavity size. Excavator should be deepened in the tooth axis direction when working in the mantle dentine. In the parapulpar dentine the excavator should be deepened in a horizontal direction. Observance of these rules prevents spontaneous pulp exposing. More dense layers of affected dentine are removed by a round or reverse conical bur, better - hard-alloy, with a mechanical hand piece. During treatment of acute or chronic moderate caries in deciduous teeth, changed dentine of the walls and the bottom of the carious cavity should be carefully removed up to unchanged in color, dense dentine. The carious cavity shape should ensure the best fixation of the filling. Special features of the carious cavity formation depend on the deciduous tooth's development stage, its localization, depth and materials used for permanent filling.

New modem composite materials, glass-ionomer cements and adhesive systems, allow wide usage of a biological expediency method for carious cavity preparation in deciduous teeth. Lukomskiy's preparation method assumes removal only of the carious dental tissues. Preservation of healthy dental tissues, minimal dental invasion and decreased number of filling materials used are advantages of the method. Though, it has some week points as well: carious process can start on the adjacent segments of the tooth; filling can fall out as a result of faulty retention. An antiseptic treatment of a carious cavity is done before filling.

ART-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 stress 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.

Filling of carious cavities in deciduous teeth. For these purposes modern filling materials are used — glass-ionomer cements, copipomers, 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.

T*reatment of acute initial caries in permanent teeth* in children is done by the way of remineralization therapy. A successfully performed remineralization of initial caries prevents further formation of a carious cavity. It is recommended to saturate the demineralized areas with calcium and phosphate ions first; they are the basis for the enamel crystal lattice. Fluoride

medications are applied next; they decrease enamel permeability owing to formation of a superficial protective film over the enamel.

The following materials are used for remineralization therapy of acute initial caries:

- 01—0,1 % sodium fluoride solution for mouth rinsing and applications;
- 1- 2 % sodium fluoride solution for applications and electrophoresis in hard dental tissues;
- fluoride varnishes;
- 10 % solution of calcium gluconate or calcium chloride;
- 2.5 % solution of calcium glycerophosphate for applications and electrophoresis; - calciferous gels.

Remineralization therapy assumes the following manipulations: dental deposit is carefully removed from tooth surface (mechanically or with hydrogen peroxide); the surface is dried with an air stream. A cotton or gauze strip soaked in 10% solution of calcium gluconate is applied (the application is changed every 4—5 min.). After the third application of mineralizing solution the tooth surface is dried and a gauze strip soaked in 2—4% sodium fluoride solution is applied for 1-3 min. The remineralization therapy course consists of 15—20 applications (daily or each other day).

In order to prolong the fluoride's action on hard tooth tissues, especially after the remineralizing therapy with calcium medications, it is advisable to use fluoride varnishes, like Fluor Protector (Vivadent), Duraphat (Colgate), Bifluorid 12 (VOCO), Belagel F, Belak F (VladMiVa), Ftorlak (Stoma). They are applied on the carefully dried enamel surface. After the procedure the patient should keep from eating or rinsing mouth for 2 hours.

The method of deep enamel fluoridation (by A. Knapwost, 2001) is used for enamel remineralization in case of acute initial caries, and implies consistent treatment of enamel with a solution of magnesium-fluoride silicate, firstly, and a suspension of highly dispersive calcium hydroxide, next. Enamel .Germetic Liquid (Humanchemie, Germany) is used for these purposes. Deep enamel fluoridation assumes the following procedures: dental deposit removal from the enamel surface; solution №1 application for 1 min; solution №2 application for 1 min. The treatment course consists of three visits. In case of high carious risk the procedure should be repeated in 1-2 weeks.

Treatment of chronic initial caries in permanent teeth. Remineralization therapy is not effective and it is not applied in these cases. In case of a significant affected areas, their preparation and filling should be performed immediately, before the carious cavity deepens. In case of small area of enamel affection a dynamic supervision without invasion, or the defect grinding followed by fluorization, are possible.

Treatment of superficial and moderate caries in permanent teeth.

Superficial and moderate caries in permanent teeth in children are treated by the way of preparation and filling. During preparation it is required to completely remove all changed (softened and pigmented) dentine from the carious cavity's walls and bottom until hard and unchanged dental tissues are revealed. The quality of carious dentine removal can be objectively estimated by special coloring dyes. Superficial and moderate caries lesions are localized on oclusal surface in fissures (or its parts) of permanent molars. Thus, for their treatment it is advisable to use a modern, minimal invasive method of preventive filling.

In case of larger carious lesion of hard tooth tissues, carious cavity prepation followed by restoration of the tooth anatomical shape with filling materials is the main method of treatment dental caries. Carious cavity preparation includes next steps:

- carious cavity opening and expanding;
- necretomy of carious hard tooth tissues;
- cavity formation;
- finishing of the cavity enamel edges.

Treatment of acute deep caries in permanent teeth. Treatment of acute deep caries in permanent teeth in children is performed in one or two visits. That depends on carious process peculiarities, tooth's formation degree, medical cavity liners that are used, cariogenic situation in the oral cavity.

In the following cases treatment of acute deep caries in permanent teeth is performed in one visit:

– acute deep caries of permanent tooth with mature roots (III-V stage of the root formation proved by the X-ray);

– compensate and sub-compensate carious activity degree in a patient;

– carious dentine on the carious cavity bottom is moderately softened, the carious cavity sides are hard after preparation;

– cariogenic situation in the oral cavity is moderate.

An incomplete removal of softened dentine of the carious cavity's bottom is a distinctive feature for the acute deep carious cavity preparation. However, this does not mean that the bottom is not prepared at all. Bottom preparation should be gently performed with a dental excavator and a mechanic handpiece with a big round bur at low speed (10—20 thousand RPM); that would prevent incidental pulp denudation. Carious cavity's sides should be carefully prepared until hard unchanged dentine. The preparation should be followed by the carious cavity's isolation from saliva, its antiseptic treatment and drying. A therapeutic cavity liner should be applied on the cavity bottom; the liner stimulates remineralization of softened dentine and production of the reparative dentine. Medical materials with calcium hydroxide are mostly often used for these purposes.

Calcium hydroxide causes an anti-inflammatory action resulting from neutralization of acid environment in the inflammatory process. High concentration of hydroxyl ions ensures its bactericidal and bacteriostatic action. During treatment of acute deep caries in one visit it is advisable to use hardening medical liners with calcium hydroxide: Dycal (Dent Splay), Life (Kerr), Recal (Great Britain), Calcimol (VOCO), etc. The calcium hydroxide liner is covered by an isolating liner of glassionomer or zinc-phosphate cements. Caries treatment is completed by applying a permanent filling from materials high adhesion.

Treatment of acute deep caries in permanent teeth in children is performed in two visits in following cases:

– caries in teeth with unformed roots (I—II root formation stage, X-ray proved);

– significant dentine softening on the bottom and walls of a carious cavity (it is impossible to prepare walls until the hard dentine layer);

– decompensated carious activity degree in a child;

– significantly expressed cariogenic situation in the oral cavity.

Carious cavity preparation and its antiseptic treatment are performed in
the first visit. The carious cavity bottom is covered with a thin layer of a non-
hardening calcium hydroxide liner — Calcicur (VOCO), Calcipulpe(Septodont); a layer of hardening calcium hydroxide is applied next - Calcimol (VOCO), Calcimol LC (VOCO), Septocalcine Ultra (Ultradent).

Treatment of chronic deep caries in permanent teeth in children is not complicated as the pulp is effectively protected with a reparative dentine layer. It is acceptable to leave pigmented hard dentine on the carious cavity bottom; though the carious cavity walls should be prepared until healthy dentine. No special medical cavity liners are used in this case. The bottom is covered with an isolating liner of zinc-phosphate or glassionomer cements. The treatment is completed in one visit with a permanent filling.

Comprehension control

1. Peculiarities of the clinical course of caries of the primary and permanent teeth with unformed
rootsin children.
2. Peculiarities of the clinical course of the primary and permanent teeth with formed roots.
3. Zinc-phosphate, silicate and silicate-phosphate cements, its composition, properties, indications for
use in pediatric dentistry.
4. Glass-ionomer cements, its composition and indications for use.

5. Compomers, its characteristics and indications for use.
6. Classification. properties, composition and indications for use of the composite filling materials
inpediatric therapeutic dentistry.
7. Amalgam, its composition, properties, indications for use.
8. General caries treatment. Indications and methods of conduction.

Test control

1. What isolating liner is recommended for filling prior to silver amalgam ?
A. Zinc phosphaste cement
B. paste containing Ca(OH)2
C. Zinc oxide eugenol paste
D. Polycarboxylate cement
E. no necessity to use liner

2. During an examination of a 9-year-old child on the cervical surface of the 12, 11, 21 and 22 teeth white spots are found which 2 weeks ago appeared. The spots are without brilliance, and can be colored with methylene. The affected teeth do not react to cold irritants. What should the dentist's tactic be to the affected teeth?
A. Remineralization therapy
B. Filling of the carious cavity
C. Polishing of the damaged areas
D. Impregnation therapy
E. Regular medical check-ups

3. During the preventive examination of 18-year-old patient white painless spots were discovered after the removal of dental deposits on the gingival part of vestibular surface of the 22 and 41 teeth. The test of enamel resistance is 1 mark. What morphological changes are characteristic to this disease?
A. Sub superficial demineralization of enamel
B. Superficial demineralization of enamel
C. Degenerative changes of odontoblast
D. Changes in a cover dentine
E. Damage of dento-enamel junction

4. A 4 -year- old child, practically healthy, has been examined with the purpose of prevention. Objectively: on the masticatory surface of the 75 tooth there is a carious cavity within the cover dentine, filled up with softened dentine. Dentoenamel junction is tender to probing. What is the best material for a permanent filling?
A. Glass ionomer cement
B. Composite resin material
C. Phosphate cement
D. Silicate cement
E. Silicate phosphate cement

5. A young man, 17 years old, complains of the presence of cosmetic defect in the form of light spots on teeth. The presence of fluorine in drinking-water is 1 mg/1. Objectively: on the

vestibular surface of the 11, 12, 21, 22 teeth; the cusps of the 16, 26, 36, 46 teeth there are white spots with a glossy surface that has existed from the moment of the eruption of the teeth. What is the most probable diagnosis?

A. Systemic enamel hypoplasia
B. Endemic fluorosis
C. Enamel erosion
D. Multiple caries
E. Amelogenesis imperfecta

6. The parents of a 2,5-year-old child complain of the front teeth decay in the maxilla which has lasted during a few months. On the contact and vestibular surfaces of the 52,51,61,62 teeth carious cavities are found within the cover dentine, filled up with softened pigmented dentine which is easily removed with an excavator. Define a provisional diagnosis.

A. Acute medium caries
B. Chronic superficial caries
C. Chronic deep caries
D. Acute deep caries
E. Chronic medium caries

7. During filling the II class according to Black carious cavities in the 36 tooth the dentist decided to use the "open sandwich" method. Which of glassionomer cements should be applied to substitute for dentine?

A. Vitremer TC (3M)
B. Vitrebond (3M)
C. Base Line (Dentsply)
D. Aqua-Cem (Dentsply)
E. Aqua-Jonobond (VOCO)

8. A 17-year- old girl, complains of the sensitivity of the teeth to sweet and sour. Objectively: on the cervical surface of the 14,13,23,24 teeth there are opaque white spots, painless to probing. The spots are stained by 2% solution of methylene blue. EOD=4 mkA. What is the most probable diagnosis?

A. Acute initial caries
B. Enamel erosion
C. Acute superficial caries
D. Enamel hypoplasia
E. Fluorosis, maculosus form

9. The parents of a 9-year-old child complain of a cosmetic defect of the right upper front tooth which erupted with defected enamel. The case history has a record of premature extraction of the 62 tooth due to the complicated caries. On the vestibular surface of the tooth there is a yellow spot with clear margins. The enamel above it did not loose brilliance. At probing no roughness is revealed. Make a provisional diagnosis.

A. Local enamel hypoplasia
B. Chronic superficial caries
C. Systemic enamel hypoplasia

D. Acute superficial caries
E. Fluorosis

10. A 13-year- old child, has been complaining of pain from cold in the lower left molar for a few months. On the masticatory surface of the 37 tooth a carious cavity was found. It has overhanging edges of enamel, located within the parapulpar dentine, filled with the light softened dentine. Probing of the bottom is painful. A short-lived pain occurs as a reaction to cold. Define provisional diagnosis.

A. Acute deep caries
B. Acute medium caries
C. Chronic fibrous pulpitis
D. Chronic medium caries
E. Chronic deep caries

Recommended literature

1. Paediatric dentistry/ Richard Welbury, Monty Duggal – 3rd ed., 2005 Copyright.
2. L.A. Khomenko. Pediatric Therapeutic Dentistry.- K.:Book-plus, 2012

PRACTICAL CLASS № 4

Theme: Clinical course, diagnosis, and differential diagnosis of pulpitis of primary and permanent teeth in children. The choice of the method of treatment depending on the stage of development.

Objective: Learn the clinical course of pulpitis of primary and permanent teeth in children, differential diagnosis and choice of the treatment method.

Pre-study test questions

1. Peculiarities of the anatomical structure of the primary teeth.
2. Peculiarities of the anatomical structure of the permanent teeth in children.
3. Topography of the root canals of the primary and permanent teeth in children.
4. Terms of formation of the roots of the permanent teeth.
5. Peculiarities of the apical foramen of the primary teeth.
6. Pulp, its definition and functions.
7. Pulpitis, etiology and pathogenesis.
8. Classification of the pulpitis.

Contents of the class

Infection is the most common cause of pulpitis. Microorganisms (predominantly streptococci, staphylococci, and mixed microflora) penetrate into the pulp chamber from the carious cavity. Mechanical trauma is the second leading cause of pulpitis. It occurs in case of concussion, fractures and during carious cavity preparation. It has been proved that the largest damages of pulp occur at speed of bur in the range of 3000-30000 rotations per minute without bur cooling. Bur speed of more than 200000 rotations per minute with the use of bur cooling is the most secure speed for preparation. Excessive dentine drying causes its dehydration and damages of pulp.

Classification of pulpitis in deciduous teeth made by the department of pediatric and preventive dentistry of the National O.O. Bogomolets Medical University:

I. Acute pulpitis (pulpitis acuta):
1) Acute serous diffuse pulpitis (pulpitis acuta serosa diffusa);
2) Acute purulent pulpitis (pulpitis acuta purulenta);
3) Acute traumatic pulpitis (pulpitis acuta traumatica).
II. Chronic pulpitis (pulpitis chronica):
1) Chronic fibrous pulpitis (pulpitis chronica fibrosa, seu simplex);
2) Chronic hypertrophic pulpitis (pulpitis chronica hypertrophica);
3) Chronic gangrenous pulpitis (pulpitis chronica gangraenosa).
III. Aggravated chronic pulpitis.
IV. Pulpitis complicated with periodontitis.

Pulpitis in deciduous teeth are extremely difficult to diagnose. As a rule children can not clearly describe their subjective sensations and objectively estimate their reaction to the doctor's diagnostic methods (percussion, probing, thermometry). The patient's reaction depends on his psycho-emotional featutres. The abovementioned is proved by a significant number (74—88 %) of divergences between the clinical and pathomorphological diagnoses.

Delopment of inflammation in the pulp and clinical picture of pulpitis in desiduous teeth have a number of features:

Inflammation of the pulp often develops in case of the shallow, carious cavity; it is explained by a number of factors: dentine layer is very thin and poorly mineralized in deciduous teeth, especially at the stage of unfinished root formation; the secondary dentine is absent; pulp horns arc located close enough to the occlusal surface; dentinal canaliculi are wide and straight; microorganisms and their toxins can easily penetrate li om the carious cavity to the pulp chamber.

- Topographic-anatomical and histological characteristics of pulp in deciduous teeth contribute to rapid spread of inflammation to the entire coronal and root pulp (within 2-3 hours).
- Chronic forms of pulpitis prevail. They develop as a result of acute pulpitis, or as a primarily chronic process tending to aggravation.
- Clinical picture of the same form of pulpitis may manifest with various symptomatic complexes depending on the stage of tooth development (unformed root, root stabilization or root resorption).
- Serous forms of pulpitis rapidly transform into the purulent ones.
- Acute forms of pulpitis (at the stage of unformed root, in particular) develop with the symptoms of perifocal periodontitis; that is explained by topographic-anatomical and histological characteristics of periodontal ligament which contribute to entering of virulent infection, toxins and products of necrosis of pulp tissue into the periodontal ligament.

Treatment of pulpitis in deciduous teeth in children is complicated by some psychological and emotional characteristics of patients of this age group. The main purpose of treatment of pulpitis in children is to eliminate pulp inflammation and to prevent inflammation in periodontium, jaws and soft tissues of maxillofacial area. It is necessary to ensure conditions for further development of immature roots and their physiological resorption.

There are five methods of treatment of pulpitis that are used in pediatric dentistry:

1)Conservative or biological method is aimed at preservation of vitality and functional activity of the entire pulp.

2)*Vital pulp amputation* is a method of removal of coronal pulp (after anesthesia) and preservation of vitality of the root pulp.

3)*Vital pulp extirpation* is a method of complete pulp removal under anesthesia.

4)Devital pulp amputation is a method of removal of the coronal pulp after its prior

devitalization.

5)Devital pulp extirpation is a method of complete removal of the pulp after its prior devitalization.

Devital amputation is a method of partial pulp removal (removal of coronal pari) after its prior devitalization, and mummification 1 uiTently, this method is widely used for treatment of some forms of pulpitis hi dceiduous teeth with immature roots or roots at the stage of resorption. In IIK'SC cases endodontic intervention is undesirable because of the risk of injury

or infection of periapical tissues, which could adversely affect the physiologi¬cal processes of the tooth development.

Indications for devital amputation of pulp in deciduous teeth with immature roots or roots at the stage of resorption:

— acute traumatic pulpitis (incidental pulp exposure during the preparation of the carious cavity);

— acute serous diffuse pulpitis;

— chronic fibrous pulpitis;

— chronic hypertrophic pulpitis.

Treatment technique. Devital amputation is performed in 2—3 visits.

In the first visit the doctor applies a devitalizing paste. For these purpose the following manipulations should be performed:

— partial necretomy — opening of a carious cavity and creating of conditions for fixation of the dressing;

— opening of the pulp horn (if it has not been exposed before) for better contact of the devitalizing paste with the pulp tissue;

— application of the devitalizing paste;

— application of a hermetic dressing for the paste fixation for the necessary term.

In deciduous teeth with immature roots or roots at the stage of resorption only paraformaldehyde paste should be used for pulp devitalization («Parapasta» (Chema, Polfa), «Depulpin» (VOCO), «Devipulp» (Septodont), etc.)

During the second visit the dentist performs amputation of the coronal pulp and application of antibacterial and mummifying paste over the root pulp. For these purposes he removes the dressing, opens pulp chamber of a deciduous tooth (taking into account its topography) and amputates the crown pulp. Pulp from orifices of root canals should be removed (if it is possible) with a medium-size spherical bur with a prolonged working part. After devitalization with paraformaldehyde paste the root pulp becomes a dry grey cord which does not react to mechanical irritants. In case of incomplete devitalization (bleeding, painful sensations of root pulp from probing) it is advisable to re-apply the devitalizing paste for 4-5 days.

Alter the amputation of coronal pulp a paste with antibacterial and dehydrating properties should be applied on the root pulp. Pastes containing formalin, paraformaldehyde, cresol, thymol and other antiseptic agents are used for these purposes. Some ready-to-use pastes can also be applied: Tepasta (Chema Polfa), Mummifying Pasta (PD, Switzerland), pastes based on resorcinol and formalin (resorcinol-formalin paste, Foredent (Spofa Dental), Forphenan Septodont).

Taking into consideration some negative properties of strong in above-mentioned pastes a zinc-eugenol paste with nil iseptics (thymol, iodoformium) is used to cover the root pulp.

During the third visit a temporary filling (if it was made in the second visit) should be replaced with a permanent one.

Devital extirpation is a complete removal of pulp after its prior devitalization.

Indications for devital pulp extirpation in deciduous teeth:

-all forms of acute and chronic pulpitis in teeth with mature roots;

-acute purulent pulpitis, chronic gangrenous pulpitis, pulpitis with clinical and radiological features of affection of periodontium — in teeth with immature roots.

*Treatment technique.*Devital extirpation is performed in 2—3 visits.

In the first visit the doctor performs partial preparation of carious cavity, disclosure of pulp chamber and application of devitalizing paste. In deciduous teeth a paraformaldehyde paste is used for pulp devitalization. The paste is applied under a hermetic dressing for 10—14 days.

During the second visit the dentist removes the dressing, opens the pulp chamber (taking into consideration its topography), and extirpates pulp from root canals. For devital extirpation of pulp in deciduous teeth with immature roots it is necessary to make a radiogram and define the stage of root formation before starting the treatment. After pulp has been removed it is necessary to fill mot canals of the deciduous tooth (up to the apical foramen) with pastes which would not irritate periodontium.

The following materials are used for filling of root canals in deciduous teeth after the devital extirpation:

1)zinc-eugenol paste, prepared ex tempore with the addition of radiocontrast agents;

2)zinc-oxide-eugenol cements (Cariosan (Spofa Dental), IRM (DentSply), Endobtur (Septodont) etc.);

3)pastes with iodoform, thymol - Iodent (VladMiVa, Russia), 5% iodoform paste, mixed ex tempore, Tempophor (Septodont), Timophorm (Alpha Beta).

Selection of filling materials for root canals in deciduous teeh depends on the stage of tooth development. For treatment of deciduous teeth with immature roots with a method of devital extirpation it is advisable to use zinc- eugenol paste mixed ex tempore. It contributes to apexification of roots in deciduous tooth. The abovementioned filling materials can be used for filling of root canals of deciduous teeth at the stage of root stabilization. For filling of root canals of deciduous teeth at the stage of root resorption it is advisable to use quickly resorpting non-hardening pastes - iodoform and thimol pastes prepared ex tempore basing on paraffinic, glycerine or camphor oils.

Treatment of pulpitis of permanent teeth in children.

Vital amputation is a method of treatment of pulpitis presupposing removal of the coronal part of pulp under anesthesia and preservation of vitality and functionality of the root pulp. In pediatric therapeutic stomatology this method is the most frequently used for treatment of permanent teeth with unformed roots, as it allows to preserve the functional activity of root pulp, and thus, it provides conditions for the growth and physiological formation of roots in permanent teeth – apexogenesis.

Indications for vital pulp amputation in permanent teeth with immature roots:

- acute traumatic pulpitis (if more than 6 hours passed since the injury and the pulp is significantly exposed);
- if treatment with biological method is ineffective or contraindicated;
- acute serous localized pulpitis;
- acute serous diffuse pulpitis (without the expressed reaction of periodontium).

The child's general somatic health state (healthy, practically healthy) and level of caries activity (compensate form) are taken into account for the choice of the method of vital amputation.

After vital pulp amputation in tooth with immature roots, the root continue to grow in length; the apical part and periodontium continue their formation. A hard tissue barrier - a dentin bridge - forms over the wound surface.

Treatment technique. Vital amputation is performed in one visit.

Treatment steps

Step 1 - local anesthesia.

Step 2 - preparation of a carious cavity taking into account topography of the pulp chamber.

Step 3 - opening of the pulp chamber with sterile fissure or spherical burs.The carious cavity should be regularly irrigated with warm solutions of non-irritating antibacterial preparation. The coronal pulp is removed with a sharp excavator.

Step 4 — hemostasis of the bleeding from the pulp stump with special preparations: Racestypine (Septodont), Vasoseptin, Viscostat (Ultradent). Some authors recommend to use warm sterile physiological solution or distilled water for controlled hemostasis during the vital amputation. After that the cavity is dried with sterilecotton balls. If the bleeding does not stop in 4-5 minutes it proves the inflammation of the root pulp and the necessity of its complete removal (extirpation).

Step 5 - surface of the root pulp is covered with a soft paste containing calcium hydroxide: Calcicur (VOCO), Calxyl rot (VOCO), Calcipulpe (Septodont), Calasept RO (Nordiska), Speiko Gal (Speiko), Hypo Cal SN (Merz). A layer of hardening preparation of calcium hydroxide or liner with calcium hydroxide is applied on a nonhardening therapeutic liner.

Step **6- permanent filling of carious cavity** with the use of isolating liner (glass ionomer cement).

After treatment of pulpitis in permanent tooth by the method of vital amputation the child should be regularly observed by a dentist (dispensary observation). The first control visits is scheduled in 10-14 days, the next – in 3, 6 and 12 month. The folloving criteria are used for the evaluation of effectiveness of treatment of pulpitis: formation of the dentin bridge (defined radiologically), completion of the root development and absence of pathological changes in periodontium.

Vital extirpation is a pulpitis treatment method presupposing complete removal of pulp under anesthesia and filling of the root canals.

Indications for the vital extirpation of pulp in permanent teeth:

- in teeth with mature root: all form of acute and chronic pulpitis, if conservative methods of treatment are ineffective or contraindicated;
- in teeth with immature roots: acute purulent pulpitis; pulpitis with expressed features of perifocal or focal periodontitis; chronic gangrenous pulpitis.

It should be noted that before performing the vital pulp extirpation in a tooth with immature roots, it is nessesary to make a radiogram and define the stage of root formation and the tooth working length.

*Treatment technique.*Vital extirpation is performed in one visit.

Treatment steps:

Step 1 – local anesthesia.

Step 2 – preparation of carious cavity and opening of the pulp chamber taking into account topography of the pulp chamber;

Step 3 – pulp removal (extirpation);

Step 4- hemostasis of the root canal bleeding. Vital pulp extirpation is accompanied by bleeding of varying intensity from the root canal. The abovementioned hemoststic preparation are used for these purpose. In case of unsuccessful hemostasis a turunda with a hemostatic solution or suspense of Ca(OH)2 is inserted into the root canal. In this case the root canal is filled in the next visit.

Step 5 – filling of the root canals. The choice of the filling materials depends on the stage of root formation. For filling of root canals in permanent teeth with formed roots, gutta-percha points in combination with hardening sealers should be used.

Criteria of the correct filling after the vital extirpation: the root canal should be filled up to the physiological root apex which is 1-1.5 mm lower than the anatomical apex. The radiogram should show the filling mass 1-1.5 mm lower from the root apex.

After the vital extirpation in the permanent tooth with immature root is finished the root canal filling should be performed in 2 steps.

Step 1 – temporary obturation of the root canal within the formed part with pastes containing calcium hydroxide.

After the root canal has been filled temporary filling of glass ionomer cement is applied. It provides the necessary hermetic sealing.

Paste containing calcium hydroxide stimulates production of osteocement and osteodentine in the area of root apex; thus, they close the apical foramen and provide apexification. The use of the paste with calcium hydroxide for temporary obturation of the root canal requires dispensary observation, when the doctor estimates the state of the paste in the root canal and the dynamics of radiological changes. Fast resorption of the paste with calcium hydroxide requires its repeated application in the canal. The first repeated application of the paste should be performed in a month, further - every 2-3 months. The average treatment lasts 12-18 months. For stimulation of apexification zinc-eugenol paste may also be used, as its resorption in the root canal is very slow. Formation of the apical barrier is estimated radiologically and clinically. The apexification lasts from 6 to 24 months.

Step 2 – permanent filling of the root canal – is performed after the apical foramen is closed. Gutta-percha points in combination with sealers or hardening pastes for root canals should be used for these purposes.

Comprehension control

1. Peculiarities of the clinical course of an acute purulent pulpitis of the primary teeth.
2. Differential diagnosis of the chronic gangrenous pulpitis.
3. Chronic hypertrophic pulpitis in children.
4. Acute diffuse pulpitis of the permanent teeth in children.
5. Differential diagnosis of the chronic fibrous pulpitis of the permanent teeth in children.
6. Biological method of treatment of the pulpitis, indications and contraindications for use.
7. Requirements for medications which are used for biological method.
8. Technic of the biological method of treatment of pulpitis in children.
9. Remedies containing calcium hydroxide, types, examples, Indications for using.
10. Method of vital amputation, indications, technic. Apexogenesis.
11. Method of devital amputation, indications, technic.
12. Method of devital extirpation, indications, technic.
13. Filling materials for obturation of the root canals of the primary teeth in children.
14. Filling materials for obturation of the root canals of the permanent teeth in children.

Test control

1. A 9 years old boy complains of pain in the tooth while eating. Objectively: on the approximal surface of the 55 tooth the deep carious cavity is observed. It combines with a tooth

cavity. Probing of the junction is sharply painful, and bleeding is marked, percussion is painless. What treatment should apply in this case?

A. Devital amputation
B. Devital extirpation
C. Vital amputation
D. Vital extirpation
E. Biological method

2. A child of 15 years complains of pain from thermal stimuli in the tooth on upper jaw on a left. Objective: 26 tooth is not changed in color and the cavity is within parapulpal dentin. The cavity is opened and deep probing is painful. Determine the optimal method of treatment.

A. Vital extirpation
B. Biological method
C. Devital amputation
D. Vital amputation
E. Devital extirpation

3. During examination of the child of 5 years old the diagnosis of the acute diffuse pulpitis of the 74 tooth was set. What method of treatment is the most appropriate in this case?

A. Extirpation of the pulp
B. Vital amputation
C. Devital amputation
D. Biological method
E. Tooth extraction

4. 12- year old boy complains on the long-lasting pain of 36 tooth after eating hot food. Objectively: The crown of the tooth is grayish, deep carious cavity is filled with soften dentin. There is a connection with pulp chamber; deep probing is painful, temperature stimuli cause pain that decreases slowly. Percussion is painless. Set the diagnosis.

A. Chronic gangrenous pulpitis
B. Chronic deep caries
C. Chronic fibrous pulpitis
D. Chronic granulated periapical inflammation
E. Chronic hypertrophic pulpitis

5. During the examination of 6- year old child the carious cavity on approximal-distal surface of 84 tooth was revealed. The cavity is fulfilled with softened pigmented dentin. Probing of the flour of the cavity is painful in one dot, after necrectomy the pain and minor hemorrhage appeared. The tooth is sensitive to thermal stimuli. Percussion is painless. Set the diagnosis.

A. Chronic fibrous pulpitis
B. Acute deep caries
C. Chronic deep caries
D. Chronic periapical inflammation
E. Chronic gangrenous pulpitis

6. An 8 year old boy complains of pain in the tooth while eating. Objectively: on the approximal surface of the 55 tooth there is deep carious cavity, which is connected with tooth cavity. Probing of the junction is sharply painful, the bleeding is marked, and percussion is painless. Determine diagnosis.

A. Chronic fibrous pulpitis
B. Chronic hypertrophic pulpitis
C. Chronic gangrenous pulpitis
D. Chronic granulated periapical inflammation
E. Chronic fibrous periapical inflammation

7. An 8 year old boy complains of pain in the tooth while eating. Objectively: on the approximal surface of the 55 tooth there is a deep carious cavity, which is connected with tooth cavity. Probing of the junction is sharply painful, the bleeding is marked, percussion painless. What paste should be imposed in the first visit?

A. Paraformaldehide
B. Arsenic
C. Resorcin – formalin
D. Tymol
E. Calcium- containing

8. An 8-year-old child complains at causeless pain in the area of lower left lateral teeth. The pain increases from hot and during chewing. In the process of examination on the distal surface of the 75 tooth a defect of a sharp-edged filling is found. Interdental papillae in the area of the 75, 36 teeth are hyperemic and swollen. Choose the most probable diagnosis:

A. Acute diffuse pulpitis, complicated by periapical inflammation
B. Acute periapical inflammation
C. Acute diffuse pulpitis
D. Acute marginal periapical inflammation
E. Acute focal pulpitis

9. A child is 8 years old. There are complains of pain in the 75 tooth during eating hot food. Objectively: the 75 tooth is discolored, the carious cavity is filled up with necrotic masses of unpleasant smell, the cavity of tooth is open, probing is not painful, percussion is negative, local lymphatic nodes are enlarged. Choose the most probable diagnosis:

A. Chronic gangrenous pulpitis
B. Chronic deep caries
C. Chronic hypertrophic pulpitis
D. Chronic fibrous pulpitis
E. Exacerbation of chronic periapical inflammation

10. A child is 6 years old. The diagnosis is chronic gangrenous pulpitis of 55 tooth. Choose the method of treatment:

A. Devital pulpectomy
B. Vital pulpectomy
C. Tooth extraction
D. Biological method

E. Filling of the carious cavity

Recommended literature

1. Paediatric dentistry/ Richard Welbury, Monty Duggal – 3rd ed., 2005 Copyright.
2. L.A. Khomenko. Pediatric Therapeutic Dentistry.- K.:Book-plus, 2012

PRACTICAL CLASS № 5

Theme: Clinical course, diagnosis and differential diagnosis of periapical inflammation of primary and permanent teeth in children. Modern endodontic instruments, peculiarities of endodontic treatment in teeth with unformed root.Prognosis.

Objective: To teach the students to diagnose the different forms of the periapical inflammation of the primary and permanent teeth in children. Familiarize students with the main stages of the endodontic treatment of children, choice of the endodontic instruments and modern filling materials for root canals obturation.

Pre-study test questions

1. Peculiarities of the structure of the periapical tissues in children at different stage of root development.
2. Function of the periapical tissues.
3. Fibres and cells of the periapical tissues..
4. Blood supply and innervation of the periapical tissues..
5. Types of root resorption.
6. Classification of the endodontic instruments.
7. Medications for the processing of the root canals.
8. Stages of the endodontic treatment.
9. Classification of the materials for the obturation of the root calans.

Contents of the class

Definition: **periapical inflammation (apical periodontitis)** is usually due to spread of infection following death of the pulp.

Etiology. The nature and behavior of lesions that form at the apex of the tooth are a reflection of the conditions that lead to the destruction of the pulp of the associated tooth.

Causes of apical periodontitis:

- infection
- trauma
- chemical irritation
- immune factor/ immunifaction.

I. Infection

1. Dental caries: Dental plaque → Dental caries →Pulpitis → Apical periodontitis

2. Periodontal pocket: the necrotic pulp probably becomes infected by bacteria from the gingival margins, leading to apical periodontitis.

3. Systemic infection (rare).

II. Trauma

1. The pulp sometimes dies from a blow which damage the apical vessels.

2. during endodontic treatment, instruments may be pushed through the apex or side of the root, damaging the periodontal membrane and carryng infected debris from the pulp chamber into the wound.

3. A high filling or biting suddenly on a hard object, sometimes caused an acute but usually transient periodontitis.

4. Oclusal trauma and orthodontic lead to trauma.

III. Chemical irritation:

1. irritant antiseptics used to sterilize a root canal can escape through the apex and damage the surrounding tissue.

2. A root-canal filling may also extend beyond the apex with similar effect.

3. Devital materials.

Clinic of the periapical inflammation of the primary teeth. The most often the chronic granulating periapical inflammation and the exacerbation of the chronic periapical inflammation are observed in the primary teeth in children.

Clinics. In most cases the pathological process is characterized by the absence of pain symptoms. The child primarily complains for the presence of a fistula (possibly with pus allocation), a carious cavity and the tooth's color change. In case of chronic granulating periapical inflammation the carious cavity is located within the parapulpar dentine. Though, it can also be located in the mantle dentine. These clinical features are caused by an acute course of caries in deciduous teeth and imperfect protective function of pulp (during the root growth and resorption periods in particular). That leads to rapid infection spread in periodotium.

Probing of the carious cavity bottom is painless in case of chronic granulating periapical inflammation. Reaction to thermal irritation is absent; tooth percussion is painless or slightly painful. Absence of pain during preparation of the enamel-dentine junction indicates on pulp destruction and development of the inflammation process in periodontium. Probing of the carious cavity bottom, its connection with the pulp chamber and orifices of root canals is painless in case of periapical inflammation in deciduous teeth. Sometimes, probing may be accompanied by insignificant pain and bleeding as a result of granulating tissue ingrowth into root canals and pulp chamber, especially during the root growth and resorption periods.

In most cases a fistula with growing granulations and purulent excretion is defined on the gingival mucosa in the projection of root apexes or bifurcation of the affected tooth. If there is no fistula, the gingival mucosa is pastose and it has a cyanotic coloring in the sick tooth area.

A destructed alveolar cortical plate and an enlightenment of bone tissue with indistinct contours are defined radiologically in the area of molars' bifurcation and roots apices. Pathological tooth resorption and perforation of the pulp chamber bottom in the bifurcation area are often observed. Destruction of a cortical plate in a permanent tooth follicle occurs in case of the pathological process extension to the permanent tooth germ.

Chronic granulating periodontitis in deciduous teeth should be differentiated with the following diseases:

— chronic moderate caries; it is characterized by pain during preparation of the enamel-dentine junction;

— chronic fibrous and gangrenous pulpitis; in this case probing of an exposed pulp horn and root canal orifices is accompanied with an acute pain reaction; there are no radiological changes;

— pulpitis, complicated with a focal periodontitis; probing of a disclosed pulp horn provokes

acute pain and bleeding.

In case of chronic periapical inflammation segments of bone tissue destruction in the periapical and in the bifurcation area are defined radiologically.

There are next differential-diagnostic features of chronic granulating periapical inflammation: presence of a fistula with purulent excretion and grown granulations on the swell, hyperemic gingival mucosa in the area of a pathological process; destructive changes in the periapical and bifurcation area of the affected tooth (radiologically defined), and the absence of pain during preparation of the enamel-dentine junction.

Chronic fibrous periapical inflammation is practically not diagnosed in deciduous teeth.

Chronic granulomatous periapical inflammation is very rare in deciduous teeth. It may develop in the root stabilization period of deciduous tooth development.

Aggravation of chronic periapical inflammation in deciduous teeth is the second frequent disease.

Clinics. Patients complain on a constant pain which increases gradually, especially during biting on the causative tooth.

Children refuse food. In case of purulent inflammation and acute periosteal reaction the patients' general condition worsens rapidly due to fever and general intoxication. Parents notice the following features: facial skin paleness; weakness; headache; disturbed sleep and appetite. An objective examination defines a carious cavity of varying sizes or a filling in the causative tooth. Pulp chamber can be closed or exposed. A purulent exudate can appear during the cavity exposure. The tooth is mobile due to the exudate accumulion in periodontium.

The gingival mucosa in the affected tooth area is hyperemic, swell and painful during palpation. In case of periosteal reaction development, smoothness of a mucobuccal fold is defined near the causative tooth and the adjacent teeth; the fold is painful during palpation. Sometimes a scar from fistula can be noticed on the modified mucosa. Regional lymph nodes are enlarged, dense, and painful during palpation.

Radiologically bone tissue destruction area with indistinct contours can be detected in periapical and bifurcation areas in case of aggravation of chronic periodontitis.

Aggravation of chronic periodontitis in deciduous teeth should be differentiated with an acute diffuse pulpitis complicated with a perifocal periodontitis. In case of the second one the tooth reacts on thermal irritation; pulp chamber is exposed and accompanied with an acute pain and bleeding; the radiogram shows destructive changes in periodontium.

Acute toxic periapical inflammation in deciduous teeth can develop as a result of an arsenic paste application for pulp devitalization, or the use of strong antiseptics of phenol group (phenol, camphorated phenol, tricresol, pheresol, resorcin and aldehydes (formalin) for the root canals obturation, especially during the root growth/resorption periods.

Acute traumatic periapical inflammation in deciduous teeth may result from an acute injury (bruise, blow), as well from the errors made by a dentist during endodontic manipulations.

Acute infectious periapical inflammation develops as a perifocal process in periodontium in case of serous or purulent diffuse pulpitis in deciduous teeth.

Clinical manifestation of acute periodontitis and aggrevated chronic periapical inflammation in deciduous teeth are very similar. Patients complain of a continuous pain in a causative tooth; the pain reinforces at biting or touching it with a tongue. The tooth may be intact in case of an acute trauma or it may have a carious cavity. In case of acute toxic periodontitis pulp chamber is partially or completely disclosed. Acute pain from vertical percussion is the main clinical feature.

The gingival mucosa in the causative tooth area is swell and hyperemic. Features of regional

lymphadenitis are not defined in most patients; however, there is insignificant hyperadenosis and soreness at palpation in some children.

There are no radiological changes in periodontium. Acute periodontitis should be differentiated with the aggravation of a chronic periodontitis, basing on the history data, as well as the radiological examination results (presence of destructive changes in periodontium and bone tissues).

Chronic periapical periapical inflammation can lead to the following complications:

- expansion of the pathological process on the permanent tooth follicle which can cause its death;
- infication of the permanent tooth follicle on the early stages of its mineralization can cause the local enamel hypoplasia formation;
- spreading of the inflammatory process on the follicle can cause its death, and as the result the sequestration of follicle can occur;
- the long lasting chronic periapical inflammation can lead to the changes of the permanent tooth follicle location which clinically is observed as oral or vestibular tooth location after the tooth eruption or torsivertion;
- destroying of the bone between primary tooth and permanent follicle due to expansion of the granulated tissue can cause the prematurely tooth eruption with low level of the enamel mineralization and risk of caries development;
- premature primary tooth extraction caused by chronic periapical inflammation, especially during the period of the root formation and at the beginning of their stabilization can lead to the permanent tooth retention, delaying of its eruption and formation of the orthodontic anomalies;
- expansion of the chronic inflammatory processes on the adjacent follicle in some causes follicular cyst formation.

Acute periapical inflammation 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, resor- cin) 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.

*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 does 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 pas- lose, 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 get a diffuse character rapidly, the serous inflammation phase may pass into the purulent one within a day.

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 fervescence 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 hyperemic, 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 due to collateral edema of soft tissues. Submandibular lymph nodes are enlarged, dense, and painful at palpation.

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 widespresd form of chronic periodontitis in children.

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 probbing 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.

The granuloma is tightly connected 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 hone 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 I he 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 wTth 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 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).

*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.

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.

Comprehension control

1.Peculiarities of the clinical course and treatment of the chronic granulating periapical inflammation of the temporary teeth and its aggravation.

2. Peculiarities of the clinical course and treatment of the acute periapical inflammation of the permanent teeth.

3. Peculiarities of the clinical course and treatment of the chronic periapical inflammation of the permanent teeth.

4. Possible complications due to periapical inflammations of the primary teeth.

5. Influence of the periapical inflammation on the body of the child.

6. Use of the physiotherapy for treatment of the periapical inflammations.

7. Indications for extraction of the primary teeth.

Test control

1.A 7.5 year-old child complains of discoloration of the 12 tooth. Objectively: the 12 tooth is sealed, gray, his percussion is painless. Rtg: at the root apex of the 12 tooth the bone is destructed like flame of fire with a clear borders. After the necrectomy, mechanical and pharmacological treatment of root canals, temporary obturation of root with calcium hydroxide (Calasept) was held. A month later replacement of calcium hydroxid was conducted . What time after of conduction radiological control can be possible to determine the formation of dense apical bridge?

A. 3-6 months
B. 1 month
C. over 1 year
D. 1 month
E. 2 months

2. 13-year-old patient complains of sharp pain in 36 tooth, especially during chewing. Five days ago the arsenic paste was imposed in 36 tooth. The child didn't appeal to dentist in time. On examination - the dress is saved in tooth, reaction to cold is absent, X-ray changes in the periodontium weren't found. Which drug should be used to eliminate this complication?

A. Unitiol
B. Chlorhexidine
C. furacilin
D. sodium hypochlorite
E. Chloramine

3. In a 14-year-old patient it was diagnosed a chronic fibrotic periodontitis of the 11 tooth . The preparation, instrumental and medicamentous treatment of canals of the 11 tooth were conducted. How should be conducted obturation of the canals?

A. By X-ray top
B. Upto 0.5 cm to the top
C. By the anatomic apex
D. Over the top
E. In physiological top

4. In a 15-year-old patient a chronic granulating periapical inflammation of the 26 tooth was diagnosed . It was decided to use a conservative method of treatment. Which medications should be used in mechanical expanding of root canal?

A. EDTA and sodium hypochlorite
B. Sodium hypochlorite and hydrogen peroxide
C. Hydrogen peroxide and chlorhexidine

D. Sodium hypochlorite and chloramine
E. Chloramine and Hydrogen Peroxide

5. A 10-year-old child was treated on acute serous periodontitis of the 31 tooth. Which material will you choose for obturation of the 31 tooth?
A. Seal Apex with gutta-percha pin
B. Resorcin - formalin paste
C. Endoform
D. Phosphate – Cement
E. Paratsyn

6. An 8-year-old child complains of discomfort in the lower left tooth and prolonged pain in the tooth during consuming hot food, bad breath. Objectively: a carious cavity of the 75 tooth which is connected with tooth chamber was found. Deep probing is sharply painful and accompanied by bleeding. Percussion is not painful. Define a preliminary diagnosis
A. Chronic gangrenous pulpitis
B. Chronic fibrous periapical inflammation
C. Chronic hypertrophic pulpitis
D. Chronic fibrous pulpitis
E. Chronic granulating periapical inflammation

7. To prevent perforation of the wall or bottom of the coronal cavity the docor have to
A. taking into account the topography of the tooth cavity
B. regular recapitulation of instrument
C. exact working length
D. root canal irrigation
E. there is no correct answer

8. Breaking of the wall in the process of uncovering the tooth cavity is a result of
A. excessive pressure on the tooth by using of the bur
B. lack of depth control of the bur
C. no correct answer
D. not filled the root canal
E. of using an aggressive instruments

9. During root canal treatment arise the following complications (choose the wrong answer):
A. breaking of the wall
B. blockage of the lumen of the root canal dentin them sawdust
C. excessive expansion of the root canal without changing its shape
D. excessive expansion of the root canal in the middle third of the inner curvature
E. change in the shape and placement of the root canal apical opening

10. Sign of the blockade of lumen root canal dentin sawdust are:
A. impossibility of introducing small size tool to the entire working length
B. free movement of large instruments
C. possibility of introducing a small size tool to the entire working length
D. pain when administered endodontic instrument
E. occurrence of bleeding from the root canal

Recommended literature

1. Paediatric dentistry/ Richard Welbury, Monty Duggal – 3rd ed., 2005 Copyright.
2. L.A. Khomenko. Pediatric Therapeutic Dentistry.- K.:Book-plus, 2012

PRACTICAL CLASS № 6-7

Theme: Periodontal diseases in children. Gingivitis, periodontitis, periodontal syndrome.Etiology, pathogenesis, clinical course, diagnosis, differential diagnosis.Main principles of periodontal diseases treatment in children.The choice of the medical remedies.

Objective: To learn with students the etiology of the gingivitis, periodontitis and periodontal syndrome in children, its clinical forms and differential diagnosis.

Pre-study test questions

1. Structure of the periodontium.
2. Anatomical and physiological peculiarities of the structure of the periodontium in different age periods.
3. Roentgenological picture of the healthy periodontium.
4. Role of the local and general factors in development of the periodontal diseases.
5. Methods of the diagnosis of the periodontal diseases in children.
6. Index assessment of the periodontal tissues in children.

Contents of the class

Periodontal diseases can occur in children. If group of infections are affecting only the gums, then they are known as gingival diseases and if supporting of tissues (Periodontal ligament, alveolar bone, cementum) of teeth are also involved along with gums, then they are known as periodontal gum diseases. Gingivitis occurs in almost all the kids and adolescents. Chances of advanced periodontal gum problems are less in children but can occur. Main cause of gum diseases in children is plaque deposits in relation to teeth which can occur because of poor oral hygiene in kid. Bleeding of gums during tooth brushing or flossing, swelling or puffiness of gums, recession of gums and bad breath are the main signs and symptoms of gum diseases in children.

Classification of the periodontal diseases:

I. Gingivitis – inflammation of the gingival mucosa without affection of the dentogingival junction.

Forms: catarrhal, hypertrophic, ulcerative;

Course: acute, chronic, aggravated, remission.

Prevalence: localized, generalized.

Degree of severity: mild, moderate, severe.

II. Periodontitis- the inflammatory destructive process in periodontal tissue with disturbed integrity of dentogingival junction.

Course: chronic, aggravated, abscess, remission.

Prevalence: localized, generalized

Degree of severity: mild, moderate, severe.

III. Periodontosis – a dystrophic lesion of periodontal tissues.

Course: chronic, remission.

Prevalence: generalized

Degree of severity: mild, moderate, severe.

IV. Idiopathic diseases with progressive lysis of periodontal tissue (Papillon-Lefevre syndrome, histiocytosis, hereditary neutropenia, decompensated diabetes, achatalasia etc.)

V. Tumors and tumor-like diseases.

Risk factors of the periodontal diseases

- increase in age

- low socio-economic status
- poor education
- low dental care utilization
- poor oral hygiene levels
- smoking
- psychosocial stress and genetic factors
- pre-existing conditions

Classification based on children's ages (Page and Schroeder, 1982)

I. Pubertal periodontitis (up 12 years)
A. Localized.
B. Generalized.
II. Juvenile periodontitis (age – 13-17 years)
III. Rapidly progressing periodontitis (age – 17-35 years)
IV. Adult type periodontitis (over 35 years).

Gingivitis is an inflammatory process affecting the soft tissues surrounding the teeth. The inflammatory process does not extend into the alveolar bone, periodontal ligament, or cementum.

Catarrhal (simple) gingivitis is characterized by the red color, loss of surface stippling, puffiness and softening of the gums, easy bleeding upon probing or spontaneous bleeding and the pain during mastication and palpation.

Chronic simple gingivitis. Discomfort in gingiva**, g**ingival bleeding provoked by mechanical trauma (tooth-brushing, food impaction by biting solid foodssuch as apples) and bleeding upon probing is of great value for the earlydiagnosis of chronic gingivitis. Gingival bleeding varies in severity, duration and ease with which it is provoked. The severity of the bleeding and the case with which it is provoked depend on the intensity of inflsmmation.

In chronic inflammation gums are bluish red. At the first stage of disease it is sometimes even difficult to distinguish clinically chronic simple gingivitis and normal gingiva. Originating as a light redness the colour changes through varying shades of red, reddish blue, and deep blue with increasing chronicity of the inflammatory process.

Hypertrophic (Hyperplastic) gingivitis. The clinical signs and symptoms include gingival overgrowth in the form of a diffuse swelling of the interdental papillae, or multiple, tiny nodules on the labial of the interdental papillae of anterior teeth, or as a marginal collar or festoon of tissue around the clinical crown of the tooth. Other symptoms include moderate to acute inflammation, soreness, tenderness, and moderate (4 to 7 mm) pocket depths.

Hypertrophic (Hyperplastic) gingivitis can be classified as follows:

forms: oedematic fibrous

generalized localized

3 stages: light - gums cover 1/3 of the tooth crown

moderate - gums cover not more than 1/2 of the tooth crown

heavy - gums cover 2/3 of the tooth crown or more

Acute Necrotizing Ulcerative Gingivitis. Also called "Vincent's infection" and "Trench Mouth". Infection caused by bacteria like – Borrelia vincentii, Fusobacterium nucleatum, Prevotella intermedia and species of Treponema and Selenomona.

Necrotizing ulcerative gingivitis most often occurs as an acute disease. Its relatively mild and more persistent form is referred to as *subacute.* Recurrent disease is marked by periods of remission and exacerbation. Reference is sometimes made to *chronic* necrotizing ulcerative gingivitis. However, it is difficult to justify this designation as a separate entity because most periodontal pockets

with ulceration and destruction of gingival tissue present comparable microscopic and clinical features.

Signs and symptoms:

– Typically, interdental papillae are inflamed, edematous and hemorrhagic.

– Involved papillae show punched out, crater like necrotic areas covered by grayish pseudomembrane.

– There is a fetid odor, extreme pain and spontaneous hemorrhage.

– Associated features like lymphadenopathy, fever and malaise may also be present.

– Involvement of PDL leads to necrotizing ulcerative periodontitis.

– If infection spreads through mucosa to skin of face, then the infection is called "Cancrum oris" or Noma.

Periodontitis

– Refers to inflammation of gingival tissues in association with some loss of attachment of periodontal ligament and alveolar bone.

– Due to progressive loss of attachment, destruction of PDL and adjacent alveolar bone occurs.

– The sulcular epithelium shifts apically along the root surface, resulting in formation of periodontal pockets.

Pathogenesis:

– For more than a century, periodontitis has been associated with dental plaque. But periodontitis has been shown to be absent in patients with extensive plaque also.

– Recent evidence indicates periodontitis results not from mere presence of plaque but from changes in proportions of bacterial species in plaque.

– Chronic periodontitis is associated with Actinobacillus actinomycetecomitans, Bacteroides forsythus and Prevotella intermedia.

– The pathogenic bacteria exist inside the plaque where they are protected from host defenses.

– Here they also show increased resistance to local / systemic antibiotics.

– These bacteria then release lipopolysaccharides which bring about the release of catabolic inflammatory mediators as host response.

– However, only presence of pathogenic bacteria is not sufficient to cause periodontitis.

– Other host factors like smoking, diabetes and heredity predilection are also significant in leading to periodontitis.

Classification

1. Chronic periodontitis
    - Localized
    - Generalized
2. Aggressive periodontitis
    - Localized
    - Generalized
3. Periodontitis with systemic diseases
    - Associated with hematologic disorders
    - Associated with genetic diseases
    - Diabetes mellitus
4. Necrotizing periodontal diseases
    - Necrotizing ulcerative gingivitis
    - Necrotizing ulcerative periodontitis
5. Abscesses of periodontium
    - Gingival abscess

- Periodontal abscess
- Pericoronal abscess

6. Periodontitis associated with endodontic lesions

Chronic periodontitis. A risk factor can be defined as a state or occurrence that increases the probability of an individual developing a disease. Risk factors for periodontal disease can be classified as local or general. Local factors, for example, an instanding lateral incisor, may serve to compromise local plaque control by hindering effective cleaning and resulting in **dental** plaque accumulation. On the other hand, general risk factors, such as an inherited disorder may predispose an individual to periodontal disease despite a good level of plaque control.

It is important to understand that if a child possesses a risk factor for periodontal disease, it does not necessarily follow that the child will develop the condition. Conversely, a patient may appear to have no risk factors, but the disease may develop subsequently. Bearing this in mind, risk factors (both local and general) should be considered when assessing, diagnosing, treating, and maintaining child patients with periodontal disease.

These can be grouped simply into four areas. There may be overlap between these areas.

– Malocclusions.
– Following traumatic dental injuries.
– Plaque retentive factors.
– Ectopic eruption

General risk factors for periodontal disease may have a genetic basis, with certain inherited conditions possessing periodontal manifestations (e.g. Papillon Lefevre Syndrome). The genetic conditions are dealt with previously in this chapter. There are also metabolic, haematological, and environmental risk factors within the general category. A full discussion of each is outwith the scope of this chapter, so the two most prevalent examples of general risk factors, diabetes mellitus and smoking will be discussed.

Children and adolescents can have any of the several forms of periodontitis as described in the proceedings of the 1999 International Workshop for a Classification of Periodontal Diseases and Conditions (aggressive periodontitis, chronic periodontitis, and periodontitis as a manifestation of systemic diseases). However, chronic periodontitis is more common in adults, while aggressive periodontitis may be more common in children and adolescents. The primary features of aggressive periodontitis include a history of rapid attachment and bone loss with familial aggregation. Secondary features include phagocyte abnormalities and a hyperresponsive macrophage phenotype.

Aggressive periodontitis can be localized or generalized. Localized aggressive periodontitis (LAgP) patients have interproximal attachment loss on at least two permanent first molars and incisors, with attachment loss on no more than two teeth other than first molars and incisors. Generalized aggressive periodontitis (GAgP) patients exhibit generalized interproximal attachment loss including at least three teeth that are not first molars and incisors. In young individuals, the onset of these diseases is often circumpubertal. Some investigators have found that the localized form appears to be self-limiting,while others suggest that it is not.Some patients initially diagnosed as having LAgP were found to have GAgP or to be periodontally healthy at a 6-year follow-up exam.

I. Localized aggressive periodontitis

– *Age incidence:* Begins around 11 - 13 years.
– *Sex predilection:* Nil
– *Signs and symptoms:*
– This type of disease is localized around the incisors and first molars
– Typically the oral hygiene is good and there may be mild or no gingival inflammation.
– The rate of bone loss is 5-10 times more than chronic periodontitis.
– Radiographic features: -
– Radiographs usually reveal bilaterally symmetrical vertical bone loss.
– Similar involvement is seen around anterior teeth also.

– Tooth mobility and migration is common.
– In 1/3rd of cases, disease progresses to a more generalized pattern.

II. Generalized aggressive periodontitis:

– *Age incidence:* Occurs before 30 years
– *Sex predilection:* Nil
– *Signs & symptoms:*
– Compared to localized variant, more teeth are involved.
– Bone loss is not limited to specific areas of jaws.

Primary dentition (prepubertal periodontitis)

The disease may present immediately after the teeth have erupted. In the generalized form the gingiva appear fiery red, swollen, and haemorrhagic. The tissues become hyperplastic with granular or nodular proliferations that precede gingival clefting and extensive areas of recession. Gross deposits of plaque are inevitable as the soft tissue changes make it difficult to maintain oral hygiene. The disease progresses extremely rapidly, with primary tooth loss occurring as early as 3-4 years of age. The entire dentition need not be affected, however, as the bone loss may be restricted to one arch. Children with generalized disease are susceptible to recurrent general infections, principally otitis media and upper respiratory tract infections.

Localized disease progresses more slowly than the generalized form and bone loss characteristically affects only incisor-molar teeth. Plaque levels are usually low, consequently soft tissue changes are minimal with gingivitis and proliferation involving only the marginal tissues.

The predominant micro-organisms that have been identified are aggressive periodontopathogens: *Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis, Fusobacterium nucleatum,* and *Eikenella corrodens*. This suggests that there is an infective component to the disease, although defects in the hosts' response have also been identified. Profound abnormalities in chemotaxis and phagocytosis of polymorphonuclear neutrophils and monocytes are frequently reported in these patients. These immunological defects are heritable risk factors that help to define phenotypically the disease entity. Conversely, they may also be associated with more serious and life-threatening conditions, and thus a full medical screen is indicated.

Oral hygiene instruction, scaling, and root planing should be undertaken at frequent intervals. Bacterial culturing of the pocket flora identifies specific periodontopathogens. If pathogens persist after oral debridement, an antibiotic such as metronidazole or amoxycillin (amoxicillin) should be given systemically after sensitivity testing, as a short course over 1-2 weeks. Generalized disease responds poorly to treatment. Some improvement has been achieved following a granulocyte transfusion in a patient with a defect in neutrophil function. Extraction of involved teeth has also produced an improvement in neutrophil chemotaxis, which suggests that the defect may be induced by certain organisms in the periodontal flora. Furthermore, in severe cases of generalized periodontitis, extraction of all primary teeth (and the provision of a removable prosthesis) can limit the disease to the primary dentition. Presumably, anaerobic pathogens are unable to thrive in the absence of teeth. When the permanent teeth erupt, bacterial culturing of the subgingival flora ensures that reinfection is detected early.

Mechanical plaque control. Plaque control refers to the removal of plaque from the tooth surface and gingival tissues, and prevention of new microbial growth. Effective plaque control results in resolution of gingival inflammation and is fundamentally important in all periodontal therapy. Periodontal treatment performed in the absence of plaque control is certain to fail, resulting in disease recurrence. Mechanical plaque control is performed using **toothbrushes, toothpaste** and

other cleaning aids. Plaque-control programmes should be tailored to the requirements of individual patients. Motivation of patients to change their behavioural habits is a great challenge and patients must be educated so they understand the importance of their contribution to maintaining health and preventing disease.

Chemical plaque control Chemical agents have been incorporated intomouthrinses and toothpastes with the objective ofinhibiting the formation of plaque and calculus.Antiplaque agents may also have a significant clinicaleffect of resolving an established gingivitis.

Scaling and root planing

Non-surgical management (NSM) of periodontal diseases

comprises OHI and scaling and root planning (SRP):

• scaling is the removal of plaque and calculus fromthe tooth surface

• root planing is the removal of subgingival plaque, calculus and necrotic cementum to leave a hard, smooth root surface.

SRP is generally undertaken with various hand instruments and / or ultrasonic sealers. As a result of OHI and SRP, plaque bacteria are reduced and there is resolution of the inflammatory lesion in the periodontium. This leads to shrinkage of the gingival soft tissues (as oedema resolves), increased resistance to probe tip penetration by the tissues at the base of the pocket (as inflammation resolves) and the formation of a long junctional epithelium at the base of the pocket. All these mechanisms contribute to the reduction in probing depths observed after effective NSM, although gingival shrinkage and resolution of inflammation have the most significant effects on pocket reduction. Clinical research has shown that effective OHI alone can reduce mean probing depths by approximately 0.5 mm, and SRP results in additional reductions of about 1.0-1.5 mm. There is no initial probing depth above which NSM does not confer a benefit to patients. However, root planing performed at sites with minimal or no pocketing is detrimental rather than beneficial. Root planing of shallow sites with initial probing depths <3 mm results in loss of attachment to the root surface as a result of mechanical trauma from instrumentation

Antimicrobials. The use of systemic antimicrobials in the management of periodontal disease should be restricted to the following conditions:

• severe necrotising ulcerative gingivitis

• multiple or severe periodontal abscesses with involvement of regional lymph nodes

• some cases of aggressive periodontitis.

Host modulation. The realization that the destructive host immuneinflammatory response to the presence of plaque bacteria in the periodontal pocket is the primary cause of periodontal breakdown has led to the concept of adjunctive host modulation using systemic medication.

Surgical treatment. The major limitation of closed SRP (non-surgical treatment) is that root surfaces cannot be visualized directly, and access for removal of subgingival plaque and calculus may be limited. Periodontal therapies (both surgical and non-surgical) are aimed at the removal of all plaque and calculus, and while this is seldom achieved, improvements in periodontal health are observed nonetheless. Therefore, while total elimination of causative factors is an appropriate goal for periodontitis, reduction of plaque and calculus below a certain threshold acceptable to the host may be a more realistic aim. This tips the balance between the host and bacteria in favour of the host, allowing reduction in the signs of inflammation and improvements in clinical parameters. It is typical, therefore, for patients to receive a course of non-surgical therapy and then to be monitored. For those sites that do not respond favorably to treatment (e.g. because of complicated local anatomy such as grooves or furcation involvements), then a decision may be taken to expose the area surgically for further treatment. The majority of periodontal surgery is undertaken to improve

access to the root surface for cleaning, generally via a flap procedure, although there are also several indications for specific surgical procedures too. It isfundamentally important that a high level of oral hygieneis maintained before and after surgery; surgical treatments will fail if plaque is not adequately controlled.

Other indications for periodontal surgery:

• crown lengthening to increase clinical crown length
• gingivectomy for the removal of overgrown gingivaltissues
• guided tissue regeneration (GTR) to regenerate periodontal supporting structures
• mucogingival surgery for correction of mucogingival and aesthetic defects.

Comprehension control

1. Classification of the periodontal disease.
2. Catarrhal gingivitis. Clinical course.Differential diagnosis.Treatment.
3. Hypertrophic gingivitis. Clinical course.Differential diagnosis.Treatment.
4. Necrotizing Ulcerative gingivitis. Clinical course.Differential diagnosis.Treatment.
5. Localized periodontitis. Clinical course.Differential diagnosis.Treatment.
6. Generalized periodontitis. Clinical course.Differential diagnosis.Treatment.
7. Prevention of the periodontal diseases.
8. Periodontal syndromes.
9. Periodontal conditions in patients with diabetes mellitus.
10. Periodontal conditions in patients with hereditary neutropenia.
11. Periodontal conditions in patients with Papillon-Lefevre syndrome.
12. Periodontal conditions in patients with histiocytosis.
13. Periodontal conditions in patients with immunodeficiency disorders.
14. General principles of the treatment of periodontal syndromes.

Test control

1. An 11-year-old girl complains about gingival haemorrhage during tooth brushing and eating. She has been suffering from this for a year. Gum of both upper and lower jaws is edematic and congestively hyperemic. Hygienic state of oral cavity is unsatisfactory. Bite is edge-to-edge. Roentgenological examination of periodontium revealed no pathological changes. What is the provisional diagnosis?

A. Chronic catarrhal gingivitis
B. Acute catarrhal gingivitis
C. Generalized periodontitis
D. Localized periodontitis
E. Hypertrophic gingivitis

2.The 12-years-old girl turned to the dentist for preventive dental examination. Objectively: teeth are intact, open bite. Swelling, hyperemia and cyanosis of vestibular surface of the gingival mucosa are objectively determined. Gingival papillae are increased in size, loosened, have rounded forms, easily bleed even at insignificant irritation. The increase of the gingival papillae of the frontal upper teeth is less than 1/3 of there hight. Make the preliminary diagnosis.

A. Hypertrophic gingivitis
B. Chronic catarrhal gingivitis
C. Acute catarrhal gingivitis
D. Gingival fibromatosis

E. No correct answer

3. The 12-years-old patient complains on bleeding of gums during tooth brushing during some years. The gingival mucosa in region of teeth 31, 32, 33, 41, 42, 43 is cyanotic, swelling. There is crowding of lower frontal incisors:onx-ray in the region of teeth 31, 32, 33, 41, 42, 43, The cortical lamina is determined along up whole length. Make the diagnosis.

A. Catarrhal gingivitis
B. Hypertrophic gingivitis
C. Localized periodontitis
D. Generalized periodontitis
E. No correct answer

4. A 16-year-old teenager complains of halitosis, general weakness, body temperature rises up to 37,6. These symptoms turned up 2 days ago, the boy has a history of recent angina. Objectively: oral cavity hygiene is unsatisfactory, teeth are covered with soft white deposit. Gums are hyperaemic, gingival papillae are covered with greyish coating. What is the most likely diagnosis?

A. Ulcero-necrotic gingivitis
B. Acute catarrhal gingivitis
C. Chronic catarrhal gingivitis
D. Hypertrohic gingivitis
E. Desquamative gingivitis

5. An 18-year-old patient complains of gingival enlargement, pain and haemorrhage during eating of solid food. Objectively: hyperaemia, gingival edema, hypertrophy of gingival edge up to 1/2 of crown height near the 12, 13, 14 teeth are noted. Formalin test is painless. What is the most likely diagnosis?

A. Hypertrophic gingivitis
B. Generalized II degree periodontitis, chronic course
C. Exacerbation of generalized I degree periodontitis
D. Ulcero-necrotic gingivitis
E. Catarrhal gingivitis

6. A young patient complains of gum bleeding and pain during mastication, unpleasant smell from the mouth. During the examination the hypertrophy of marginal gums in the areas of 11, 12, 13, 21, 22, 23, 34, 33, 32, 31, 41, 42, 43, 44 teeth on 1/3 of their crown’s height was found. Dental calculus and periodontal pockets of 3-4 mm of depth were present as well in mentioned areas. What is the most probable diagnosis?

A. General periodontitis of I degree
B. General periodontitis of II degree
C. Hypertrophic gingivitis, fibrous form
D. Hypertrophic gingivitis, granulated form
E. Local periodontitis of I degree

7. Preventive examination of tongue back of a 6-year-old child revealed areas of epithelium desquamation in form of red oval spots located close to the zones of hyperkeratinization of filiform

papillae. Clavate papillae are hypertrophic. There are no complaints. The child has a history of intestinal dysbacteriosis. What is the most likely diagnosis?

A. Glossitis exfoliativa
B. Candidal glossitis
C. Acute catarrhal glossitis
D. Rhomboid glossitis
E. Herpetic affection of tongue

8. In a 15 year-old patient reviled - generalized periodontitis. With what diseases is necessary to differentiate thie diagnosis?

A. With catarrhal gingivitis, periodontal syndrome in hereditary neuropenia, eosinophilic granuloma.
B. With acute catarrhal gingivitis, periodontitis marginal, papillitis
C. With catarrhal and hypertrophic gingivitis, abscess odontogenic
D. With hypertrophic gingivitis, periodontitis
E. With hypertrophic gingivitis, gingival fibromatosis, papillitis

9. A 7 month old child was brought to a dentist because of an ulcer in the oral cavity. The child was born prematurely. She has been fed with breast milk substitutes by means of a bottle with rubber nipple. Objectively: on the border between hard and soft palate there is an oval ulcer 0,8x1,0 cm large covered with yellowish-grey deposit and surrounded with a roll-like infiltration. Make a provisional diagnosis:

A. Bednar's aphtha
B. Setton's aphtha
C. Tuberculous ulcer
D. Acute candidous stomatitis
E. Acute herpetic stomatitis

10. Examination of an 11-year-old boy revealed thickened, somewhat cyanotic, dense gingival margin overlapping the crowns of all teeth by 1/2 of their height. Fedorov-Volodkina oral hygiene index is 2,6, PMA index is 20%. X-ray picture shows no pathological changes of periodontium. The child has a 2-year history of neuropsychiatric treatment for epilepsy. Make a provisional diagnosis:

A. Chronic hypertrophic gingivitis
B. Chronic catarrhal gingivitis
C. Localized periodontitis
D. Acute catarrhal gingivitis
E. Generalized periodontitis

Recommended literature

1. Paediatric dentistry/ Richard Welbury, Monty Duggal – 3rd ed., 2005 Copyright.
2. L.A. Khomenko. Pediatric Therapeutic Dentistry.- K.:Book-plus, 2012

PRACTICAL CLASS № 8

Theme: Differential diagnosis of diseases of oral mucosa in children (viral, bacterial, traumatic, allergic origin and during somatic diseases).

Objective: To learn the peculiarities of the differential diagnosis of the diseases of oral mucosa and lips in children (viral, bacterial, traumatic, allergic origin and in case of somatic diseases)

Pre-study test questions

1. Anatomical and physiological peculiarities of the structure of oral mucosa in children at different age.
2. Morpho-functional peculiarities of the structure of lips in children at different age.
3. Primary lesions of the oral mucosa of lips in children (viral, bacterial, traumatic, allergic origin and with somatic diseases).
4. Main and additional methods of diagnosis.
5. Laboratorial methods of diagnosis.
6. Allergy. Definition.Etiology.Pathogenesis.
7. Classification of the allergens.
8. Stage, types and mechanism of the allergic reaction.

Contents of the class

Measles (Rubella) occurs in children. The incidence of measles has dramatically decreased as a result of the measles vaccine; however, it remains a significant problem.

Pathophysiology

The measles virus is a paramyxovirus belonging to the Morbillivirus genus. The paramyxovirus can survive for as long as 2 hours in the air and on surfaces. Measles is spread by direct contact via droplets from respiratory tract secretions in patients who are infected. It is considered one of the most communicable infectious diseases.

The initial site of infection is the respiratory tract epithelium. Multiplication of the measles virus in the respiratory tract epithelium and regional lymph nodes is followed by a primary viremia, with spread to the reticuloendothelial system. A secondary viremia occurs upon breakdown and necrosis of the reticuloendothelial cells, and the virus infects the leukocytes. During the secondary viremia, infection may spread to the thymus, spleen, lymph nodes, liver, skin, and lungs.

Mortality/morbidity

The number of deaths caused by measles has been approximately 1-2 per 1000 cases. Young children and adults are at higher risk of death. Pneumonia accounts for approximately 60% of measles-related deaths. In children with measles, pneumonia-related deaths are most common, whereas in adults, encephalitis-related deaths are more common. Measles-related fatalities are increased in children with leukemia or HIV infection, who are immunocompromised.

Malnourishment, particularly vitamin A deficiency, is a factor that influences the severity of measles. Complications have occurred in as many as 30% of patients with measles. The complications are more severe in young children and adults. The most commonly reported complication from 1985-1992 was diarrhea, followed by otitis media and pneumonia.

In Africa, measles is the leading cause of blindness in children.

Clinical history

The incubation period for measles is the time from exposure to the prodrome. This period is approximately 10-14 days and is longer in adults than in children.

The prodrome phase lasts for several days and likely coincides with the secondary viremia phase. It is manifested by malaise, fever, anorexia, conjunctivitis, and respiratory symptoms. Toward the end of the prodrome and just prior to the appearance of the rash, Koplik spots are observed. The skin eruption of measles lasts approximately 5-6 days. The period of uncomplicated illness from the late prodrome to disappearance of skin lesions and fever lasts 7-10 days.

The fever in affected individuals can peak as high as 38-39C. Patients also experience respiratory symptoms, such as cough and coryza (runny nose), which may resemble a severe upper respiratory tract infection.

Koplik spots are pathognomonic for measles. They are located on the buccal mucosa in the premolar and/or molar area. Occasionally, in severe cases of measles, several areas of the oral cavity may be affected by the enanthem. The intraoral lesions may persist for several days and begin to slough with the onset of the rash.

Koplik spots consist of bluish-gray specks against an erythematous background. They have been compared to grains of sand. As few as 1 spot and as many as 50 spots may occur. The lesions are plaquelike or nodular and oval or round. The measles rash often begins near the hairline and then involves the face and the neck; over the next few days, it progresses to the extremities and finally to the palms and the soles. The rash is erythematous and maculopapular and may become confluent as it progresses. It lasts approximately 5 days and resolves in the same order it appeared, from the face and the neck to the extremities.

Differentials

Koplik spots - Cheek chewing keratotic lesions

Large Fordyce granules

Laboratory studies

All specimens collected for viral culture in cases suggestive of measles should be sent to the nearest state public health laboratory. The measles virus can be isolated from blood, urine, or nasopharyngeal secretions. Clinical specimens and serologic specimens should be obtained at the same time and preferably within 7 days of the onset of the rash. The easiest method that aids in confirming the diagnosis is to test for immunoglobulin M (IgM) antibody levels in a single specimen from an individual who is infected. A person who is infected or has received the vaccine initially has an IgM response followed by an immunoglobulin G (IgG) response. IgM antibodies are present for 1-2 months after exposure to the measles virus, and IgG antibodies are present for many years.

IgG tests (eg, enzyme-linked immunoabsorbent assay, hemagglutination inhibition, indirect fluorescent antibody tests) are conducted on 2 specimens. The first specimen is obtained during the acute phase (within 4 d of the start of the rash), and the second specimen is collected during the convalescent phase (2-4 wk later). Positive results show increased levels of IgG in the convalescent specimen.

Histologic findings

Oral and cutaneous lesions show necrosis of the superficial aspects of the epithelium with an inflammatory infiltrate that consists of neutrophils. Multinucleated giant cells, known as Warthin-Finkeldey cells, may be present in lymphoid tissue and lungs.

Complications

No oral complications are reported from measles. Diarrhea, otitis media, and pneumonia are the more common complications encountered from measles infection.

One case of acute encephalitis occurs in every 1000-2000 cases of measles. Acute encephalitis begins 6 days after the onset of the rash; symptoms include high-grade fever, headache, stiff neck, convulsions, and coma. The fatality rate is approximately 15%.

Subacute sclerosing panencephalitis is a previously unexplained disease that occurs months to years after the initial measles infection. Progressive deterioration of intellect, convulsive seizures, motor abnormalities, and, eventually, death characterize subacute sclerosing panencephalitis.

When measles infection occurs during pregnancy, the likelihood of early labor, spontaneous abortion, and low birth weight increases. Whether birth defects are caused by measles infection is questionable.

Prognosis

The prognosis is good in well-nourished children.

In children and adults who are immunocompetent, **primary herpetic infections** may be annoying and uncomfortable, but they rarely cause significant morbidity or mortality.

In individuals who are immunosuppressed, primary herpetic infections can be severe, and, occasionally, they can cause esophagitis, encephalitis, keratoconjunctivitis, and other diseases. The other forms of HHV can result in death. Herpes infections occasionally trigger erythema multiforme.

Age

Primary herpes infections typically occur during childhood or youth, although occasional cases are observed in older individuals. Recurrent HHV-1 infections typically occur throughout life and are particularly triggered by stress, illness, immune compromise, or other factors. Herpes zoster usually affects patients older than 40 years.

Clinical history

HHV-1

When HHV-1 infection recurs, it has different and distinct oral and perioral presentations from primary herpetic gingivostomatitis.

Primary herpes infection (primary herpetic gingivostomatitis) usually occurs in children or adolescents who have not been previously exposed to the virus. Many primary infections are asymptomatic. Symptomatic primary infection, with multiple, small, clustered vesicles in numerous locations, can occur anywhere in the oral cavity, on the perioral skin, on the pharynx, or on the genitalia. Headache, fever, painful lymphadenopathy, and malaise are common. Antibody production follows, and the virus may become latent in sensory ganglia, often the trigeminal ganglion. Primary herpetic gingivostomatitis usually resolves within approximately 14 days.

Recurrent herpes lesions are commonly referred to as cold sores. Recurrent herpes occurs in approximately one third of patients who have experienced primary herpetic gingivostomatitis. When the disease manifests extraorally, prodromal burning or itching often precedes vesicle formation. Recurrent herpes is a more limited disease than primary herpes. Unlike primary herpes, it occurs on keratinized mucosa (usually the lips, attached gingiva, and/or the hard palate). Vesicles are present in one discrete area, typically the same site every time in any given patient. Such sites include the vermilion border of the lips, the perioral skin, the hard palate, or, occasionally, the gingiva or the dorsal aspect of the tongue. Because vesicles can easily rupture intraorally, only an ulcer may be observed in some cases. Lymphadenopathy and systemic manifestations are much milder than in the primary disease.

Triggers for recurrence may include sunlight exposure, physical or emotional stress, or systemic illness for extraoral lesions and trauma (eg, a dental procedure) for intraoral lesions.

In immunosuppressed individuals, recurrent herpes lesions may occur on any oral mucosal surface, including nonkeratinized sites. They also may manifest solely as lesions on the dorsal aspect of the tongue. Such a presentation has been variously reported as red or white nodules, painful nonvesicular ulcerations, fissured, and, rarely, as a tongue mass. Herpes lesions in immunocompromised individuals are often severe. Such atypical presentations in an individual who is immunocompetent may lead the clinician to further investigate the patient's immune status.

HHV-2

HHV-2 infection is less common in the oral cavity than HHV-1 infection; however, its oral manifestations are clinically indistinguishable from HHV-1 infection. Assessment of HSV-2 shedding by polymerase chain reaction has detected oral HSV-2 shedding in the absence of an oral lesion, but concurrent with genital HSV-2 reactivation. This was more common in HIV-positive males.

HHV-3

HHV-3 is responsible for chickenpox and shingles.

Primary varicella, or chickenpox, usually occurs in children aged 3-6 years who are not immunized at the time of their first exposure to the virus. Itchy vesicles begin on the skin of the trunk and spread to the skin of the head. Intraoral and pharyngeal vesicles may occur. Antibody production follows, and the virus usually becomes latent in the dorsal root ganglia. Healthy children usually recover uneventfully, with a mortality rate of fewer than 2 deaths per 100,000 cases. However, older patients may experience more severe symptoms, and, in patients who are immunocompromised, the mortality rate may approach 18%.

Recurrent varicella, also known as herpes zoster or shingles, usually occurs in adults, and its incidence increases with age. It can occur in any patient who has had chickenpox and only rarely occurs in patients who have received chickenpox immunization. Recurrent varicella may occur when cellular immunity decreases. It results in a vesicular rash that usually affects a single dermatome. Inside the oral cavity, this may be observed as vesicles or ulcerations that stop sharply at the midline. A prodrome of pain, burning, or itching that mimics a toothache may occur.

After resolution of the rash, postherpetic neuralgia may linger for a month or longer, especially in patients who are immunosuppressed or in those older than 50 years.

Unusual complications can include devitalization of teeth, root resorption, osteonecrosis.

Ramsay-Hunt syndrome arises when the virus emerges from latency in the geniculate ganglion. It involves cranial nerve VII (facial nerve), which has both motor and sensory functions. Manifestations may include paralysis that involves the levator palati muscle and the face; hoarseness; loss of secretory function (eg, dry mouth, loss of taste); vertigo; tinnitus; pain; and vesicles involving the pharynx, the eardrum, the external ear, or the tympanic membrane.

HHV-4

HHV-4 is most commonly known as the agent that causes infectious mononucleosis, although it has been linked to African Burkitt lymphoma, other lymphoproliferative diseases, and some nasopharyngeal carcinomas.

Primary infection, infectious mononucleosis occurs on first exposure to the virus, usually during young adulthood. It is often a subclinical infection. The virus (usually acquired from infected saliva) replicates in the cells of the mucosa and salivary glands and spreads to B lymphocytes and the bloodstream. If the patient is immunocompetent, cytotoxic T cells become activated and a characteristic lymphadenopathy (notably involving the posterior cervical nodes) accompanies tonsillitis and hepatosplenomegaly. Tonsillitis may be severe and may encroach on the airway.

Thrombocytopenia may complicate the infection, and petechiae may be noted at the junction of the hard and soft palates. The patient may report headache, fever, malaise, myalgia, and fatigue.

Latent infection of B lymphocytes follows acute disease. EBV has been detected in saliva as long as 18 months after recovery from clinically evident disease.

Hairy leukoplakia, caused by EBV, primarily occurs in adults who are immunosuppressed. Hairy leukoplakia manifests as asymptomatic white lesions on the lateral border of the tongue, often bilaterally. The lesions may be observed on the adjacent dorsal or ventral surface of the tongue. Occasionally, lesions are present in other sites, such as the buccal mucosa near the commissures. The lesions have a corrugated, linear appearance and may appear granular or nodular or may have hairlike projections. Hairy leukoplakia may be the first manifestation of immunosuppression and may prompt the clinician to test the patient's HIV status. The presence of hairy leukoplakia is significantly associated with an HIV viral load of at least 3000 copies/μ L.

EBV has been detected in aggressive periodontal lesions more often than in less diseased periodontal tissues, and EBV has also been detected in periapical lesions. The significance of these findings is unclear.

HHV-5

Primary CMV infection is usually asymptomatic in patients who are immunocompetent. The virus is shed by glandular secretions, including saliva. It occasionally is shed in urine. Primary CMV infection can be asymptomatic, but it can also mimic mononucleosis. Clinical disease is more common in neonates and in patients who are immunosuppressed than in other individuals.

CMV can persist indefinitely in the host. Reactivation of latent infection can occur in patients who are immunosuppressed, including most patients who have undergone organ transplantation and as many as 90% of patients with AIDS. Latent CMV infection may cause esophagitis, which is occasionally accompanied by oral ulcerations or erythema. The disease can also affect many other body systems, including the colon, eyes, liver, lungs, or brain. The oral ulcerations are clinically nonspecific, and a biopsy is required for definitive diagnosis. A patient with HIV infection who develops CMV oral ulcerations is at high risk for progression to AIDS.

Congenital CMV infection affects 0.5-2.2% of newborns. It is frequently asymptomatic, but oral manifestations may include enamel hypoplasia of the primary teeth.

HHV-7

HHV-7 infection has been associated with roseola infantum, acute hemiplegia of childhood, respiratory tract infections, and hepatitis. It has also been linked to seizures in children with febrile illnesses. HHV-7 has been identified in the saliva of adults, and this is most likely where the virus persists chronically.

HHV-8

DNA sequences of HHV-8 have been identified in persons with KS. HHV-8, also termed KS-associated herpesvirus, may be important in causing and/or maintaining KS lesions. KS in the oral cavity follows the same disease pattern as KS in other body sites, and, initially, the lesion may appear as a red, purple, or dusky patch that enlarges into a plaque and later progresses into a tumorous mass. It is observed most frequently in immunosuppressed patients and rarely occurs in children. In the oral cavity, early KS may mimic an amalgam tattoo. The palate is the initial site of intraoral KS in approximately half the cases; other favored sites include the gingiva, the tongue, and the tonsillar area.

Diagnosis is often made based on the clinical findings alone, especially for HHV-1, HHV-2, and HHV-3. A smear of an intact viral vesicle may be helpful to confirm the clinical diagnosis. Smear results may reveal virally altered epithelial cells. Direct immunofluorescence antibody tests

and culturing help identify the causative virus. Biopsy is usually required to confirm a diagnosis of KS, and it may be required to confirm the diagnosis of other conditions.

Histologic findings

Herpes simplex infection is characterized by an acantholytic intraepidermal vesicle with epithelial giant cells. The cells exhibit nuclear molding and peripheral accentuation of the nucleoplasm. Underlying leukocytoclastic vasculitis is typically present. Zoster has similar findings, but the leukocytoclastic vasculitis is more pronounced.

CMV infection manifests as enlarged endothelial cells. The cells have ample cytoplasm and an owl's eye nucleus.

KS is typically a neoplastic spindle cell proliferation with erythrocytes in slit like spaces and extravasation between the neoplastic cells.

LEUKEMIA

Malignancy is second only to accidents as the leading cause of death in children. Leukemias are hematopoietic malignancies in which there is a proliferation of abnormal leukocytes in the bone marrow and dissemination of these cells into the peripheral blood. The abnormal leukocytes (blast cells) replace normal cells in bone marrow and accumulate in other tissues and organs of the body.

Leukemia is classified according to the morphology of the predominant abnormal white blood cells in the bone marrow. These types are further categorized as acute or chronic, depending on the clinical course and the degree of differentiation, or maturation, of the predominant abnormal cells.

Acute leukemia is the most common malignancy in children, with about 2500 new cases diagnosed annually. Thus acute leukemia accounts for about one third of all childhood malignancies; of these, approximately 80% are lymphocytic (acute lymphocytic leukemia, or ALL). Chronic leukemia in children is rare, accounting for less than 2% of all cases.

Leukemia affects about 5 in 100,000 children.The peak incidence is between 2 and 5 years of age. Although the cause of leukemia is unknown, ionizing radiation, certain chemical agents, and genetic factors have been implicated. For example, children with chromosomal abnormalities (Down syndrome and Bloom syndrome), children with an identical twin who has leukemia, and children with immunologic disorders have an increased risk of leukemia.

The clinical manifestations of acute leukemia are caused by the infiltration of leukemia cells into tissues and organs. Infiltration and proliferation of leukemia cells in the bone marrow lead to anemia, thrombocytopenia, and granulocytopenia. Because these cytopenias develop gradually, the onset of the disease is frequently insidious. The history at presentation may reveal increased irritability, lethargy, persistent fever, vague bone pain, and easy bruising. Some of the more common findings on initial physical examination are pallor, fever, tachycardia, adenopathy, hepatosplenomegaly, petechiae, cutaneous bruises, gingival bleeding, and evidence of infection.

In approximately 90% of the cases of acute leukemia aperipheral blood smear reveals anemia and thrombocytopenia. In about 65% of cases the white blood cell count is low or normal, but it may be greater than 50,000 cells/mm3.

When a new case of leukemia is diagnosed, the patient is hospitalized and therapy is directed toward stabilizing the patient physiologically, controlling hemorrhage, identifying and eliminating infection, evaluating renal and hepatic functions, and preparing the patient for chemotherapy.

These interventions proceed while the definitive studies to determine the exact type of leukemia are undertaken. These include obtaining bone marrow for microscopic analysis, special cytochemical staining, immunophenotyping by flow cytometry, and cytogenetic analysis. The goal of treatment is to induce and maintain a complete remission, which is defined as resolution of the physical findings of leukemia (e.g., adenopathy, hepatosplenomegaly, petechiae) and normalization of peripheral blood counts and bone marrow (less than 5% blasts).

The basic principle of treatment of ALL is substantially different from that of acute myelogenous or nonlymphocytic leukemia (ANLL). In general, the treatment of ANLL is very intense and results in profound bone marrow hypoplasia, but the treatment duration is usually short (less than 1 year). For ALL, the treatment is less intense but more prolonged (21/2 to 31/2 years).

In all, the treatment regimens vary considerably depending on prognostic factors and the parameters being evaluated by the strategists' cooperative group (e. g., children's oncology group). The initial phase of treatment, induction, incorporates the use of a combination of antileukemic drugs at staggered intervals during a 4-week regimen. This combination of drugs should rapidly destroy the leukemic cells, yet maintain the regenerative potential of the non-malignant hematopoietic cells within the bone marrow. About 95% of patients with ALL will be in complete remission at day 28 of therapy. The second phase of ALL treatment, consolidation, attempts to consolidate remission and intensify prophylactic central nervous system (CNS) treatment.

Prevention of CNS relapse uses intrathecally administered chemotherapy (methotrexate with or without cytosine arabinoside and hydrocortisone) to destroy leukemic cells within the CNS. This chemotherapeutic agent is instilled directly into the lumbar spinal fluid because antileukemic drugs do not readily cross the blood-brain barrier. Intensive intrathecal chemotherapy to prevent CNS relapse has replaced cranial irradiation for patients in the good and intermediate-risk groups of ALL patients. However, both cranial irradiation and intrathecal chemotherapy are still used to prevent CNS relapse in high-risk ALL patients.

The third phase of treatment, interim maintenance, uses a combination of agents that are relatively nontoxic and require only monthly visits to the outpatient clinic. In most cases, another phase, delayed intensification, follows interim maintenance. This serves to intensify antileukemic therapy again after a short period of less intensive therapy. The addition of a late phase of intensive therapy substantially improves survival in patients with ALL. Following delayed intensification, therapy continues for 2 years for girls and 3 years for boys (maintenance phase), with chemotherapeutic agents given as in interim maintenance.

The prognosis for a child with acute leukemia has improved dramatically over the past 30 years. Thirty-five years ago, there would have been little need to discuss dental treatment for a child with leukemia because the disease was invariably fatal, in most cases within 6 months of diagnosis. Today, with the development of new and better antileukemia drugs, the use of intensive combination drug therapy, the incorporation of radiation therapy, and improvements in diagnostic techniques and general supportive care, complete cures are being achieved in the majority of patients. Pre-treatment prognostic factors identify patients who are likely to benefit from either standard or more intensive therapy. Factors that identify patients who are likely to benefit from the type of therapy just outlined, which causes relatively minimal toxicity, are patient age between 1 and 10 years, a white blood cell count of less than 50,000/mm3,

lymphoblast morphology that is not of the Burkitt type, and absence of certain cytogenetic abnormalities

Children with ALL who are at particularly high risk are those who are younger than 1 year of age at diagnosis, have a high white blood cell count, or have the cytogenetic abnormalities just mentioned. More intrusive treatment regimens are used for these patients.

The prognosis for children with ANLL has improved significantly over the past several years, with event-free survivals of approximately 50% (at least 3 years) for children receiving chemotherapy and somewhat higher percentages for patients undergoing allogeneic bone marrow transplantation after achieving remission with chemotherapy. The treatment regimens are intrusive and result in profound bone marrow suppression. These patients have severe prolonged neutropenia and often have severe mucositis.

ORAL MANIFESTATIONS OF LEUKEMIA

Pathologic changes in the oral cavity as a result of leukemia occur frequently. Oral signs or symptoms suggestive of leukemia have been reported in as many as 75% of adults and 29% of children with leukemia. The lower incidence of oral manifestations in children can be attributed in part to the early age at diagnosis and the high percentage of ALL in the pediatric age group. The incidence of ALL peaks at 3 years of age, when preexisting inflammatory and degenerative changes are comparatively less frequent. Abnormalities in or around the oral cavity occur in all types of leukemia, and in all age groups. However, oral pathoses are more commonly observed in acute leukemias than in chronic forms of the disease. Oral findings suggestive of leukemia are also more common in nonlymphocytic leukemias. The most frequently reported oral abnormalities attributed to the leukemic process include regional lymphadenopathy, mucous membrane petechiae and ecchymoses, gingival bleeding, gingival hypertrophy, pallor, and nonspecific ulcerations. Manifestations seen occasionally are cranial nerve palsies, chin and lip paresthesias, odontalgia, jaw pain, loose teeth, extruded teeth, and gangrenous stomatitis. Each of these findings has been reported in all types of leukemia. Regional lymphadenopathy is the most frequently reported finding. Gingival abnormalities, including hypertrophy and bleeding, are more common in patients with nonlymphocytic leukemia, whereas petechiae and ecchymoses are more common in those with ALL.

Like the systemic manifestations of leukemia, oral changes can be attributed to anemia, granulocytopenia, and thrombocytopenia, all of which result from the replacement of normal bone marrow elements by undifferentiated blast cells, or to direct invasion of tissue by these leukemic cells. Very high circulating white blood cell numbers in the peripheral blood can lead to stasis in small vascular channels. The subsequent tissue anoxia results in areas of necrosis and ulceration that can readily become infected by opportunistic oral microorganisms in patients with neutropenia. A person with thrombocytopenia, having lost the capacity to maintain vascular integrity, is likely to bleed spontaneously. Clinical manifestations of this are petechiae or ecchymoses of the oral mucosa or frank bleeding from the gingival sulcus. The propensity for gingival bleeding is greatly increased in persons with deficient oral hygiene, because accumulated plaque and debris are significant local irritants. Direct invasion of tissue by an infiltrate of leukemiccells can produce gingival hypertrophy. Such gingival changes can occur despite excellent oral hygiene. Infiltration of leukemic cells along vascular channels can result in strangulation of pulpal tissue and spontaneous abscess formation as a result of infection or focal areas of liquefaction necrosis in the dental pulp of clinically and radiographically sound teeth. In a similar fashion, the teeth may rapidly loosen as a result of necrosis of the periodontal ligament. Skeletal lesions caused by leukemic infiltration of bone are common in

childhood leukemia. The most common finding is a generalized osteoporosis caused by enlargement of the haversian canals and Volkmann canals. Osteolytic lesions resulting from focal areas of hemorrhage and necrosis and leading to loss of trabecular bone are also common. Evidence of skeletal lesions is visible on dental radiographs in up to 63% of children with acute leukemia. Manifestations in the jaws include generalized loss of trabeculation, destruction of the crypts of developing teeth, loss of lamina dura, widening of the periodontal ligament space, and displacement of teeth and tooth buds. Because none of the oral changes is a pathognomonic sign of leukemia and all can be associated with numerous local or systemic disease processes, a diagnosis of leukemia cannot be based on oral findings alone. Such changes should, however, alert the clinician to the possibility of malignancy as the underlying cause.

Chronic Aphthous Recurrent Stomatitis.Etiology.Clinical characteristics, diagnostics, treatment.

An aphthous ulcer also known as a canker sore, is a type of oral ulcer, which presents as a painful open sore inside the mouth or upper throat characterized by a break in the mucous membrane. Its cause is unknown, but they are not contagious. The condition is also known as aphthous stomatitis, and alternatively as Sutton's Disease, especially in the case of major, multiple, or recurring ulcers.

The term aphtha means ulcer; it has been used to describe areas of ulceration on mucous membranes. Aphthous stomatitis is a condition which is characterized by recurrent discrete areas of ulceration which are almost always painful. Recurrent aphthous stomatitis (RAS) can be distinguished from other diseases with similar-appearing oral lesions, such as certain viral exanthems or herpes simplex, by their tendency to recur, and their multiplicity and chronicity. Recurrent aphthous stomatitis is one of the most common oral conditions. At least 10% of the population has it, and women are more often affected than men. About 30–40% of patients with recurrent aphthae report a family history.

Classification

Minor ulceration."Minor aphthous ulcers" indicate that the lesion size is between 3 mm (0.1 in)-10 mm (0.4 in). The appearance of the lesion is that of an erythematous halo with yellowish or grayish color. Extreme pain is the obvious characteristic of the lesion. When the ulcer is white or grayish, the ulcer will be extremely painful and the affected lip may swell. They may last about 1 week.

*Major ulcerations.*Major aphthous ulcers have the same appearance as minor ulcerations, but are greater than 10 mm in diameter and are extremely painful. They usually take more than a month to heal, and frequently leave a scar. These typically develop after puberty with frequent recurrences. They occur on movable nonkeratinizing oral surfaces, but the ulcer borders may extend onto keratinized surfaces. They may last about 10 to 14 days.

Herpetiform ulcerations

This is the most severe form. It occurs more frequently in females, and onset is often in adulthood. It is characterized by small, numerous, 1–3 mm lesions that form clusters. They typically heal in less than a month without scarring. Supportive treatment is almost always necessary.

Signs and symptoms

Aphthous ulcers usually begin with a tingling or burning sensation at the site of the future aphthous ulcer. In a few days, they often progress to form a red spot or bump, followed by an open ulcer.

The *aphthous ulcer* appears as a white or yellow oval with an inflamed red border. Sometimes a white circle or halo around the lesion can be observed. The gray-, white-, or yellow-colored area

within the red boundary is due to the formation of layers of fibrin, a protein involved in the clotting of blood. The ulcer, which itself is often extremely painful, especially when agitated, may be accompanied by a painful swelling of the lymph nodes below the jaw, which can be mistaken for toothache; another symptom is fever. A sore on the gums may be accompanied by discomfort or pain in the teeth.

Causes

The exact cause of many aphthous ulcers is unknown but citrus fruits (e.g. oranges and lemons), physical trauma, stress, lack of sleep; sudden weight loss, food allergies, immune system reactions and deficiencies in vitamin B12, iron, and folic acid may contribute to their development. Nicorandil and certain types of chemotherapy are also linked to aphthous ulcers. One recent study showed a strong correlation with allergies to cow's milk. Aphthous ulcers are a major manifestation of Behcet disease,and are also common in people with Crohn's disease.

Trauma to the mouth is the most common trigger. Physical trauma, such as that caused by toothbrush abrasions, laceration with sharp or abrasive foods (such as toast, potato chips or other objects), accidental biting (particularly common with sharp canine teeth), after losing teeth, or dental braces can cause aphthous ulcers by breaking the mucous membrane. Other factors, such as chemical irritants or thermal injury, may also lead to the development of ulcers. Using toothpaste without sodium lauryl sulfate (SLS) may reduce the frequency of aphthous ulcers but some studies have found no connection between SLS in toothpaste and aphthous ulcers. Celiac disease has been suggested as a cause of aphthous ulcers; small studies of patients (33% or 1 out of 3) with Celiac disease did demonstrate a conclusive link between the disease and aphthous ulcers vs control group (23%) but some patients benefited from eliminating gluten from their diet.

Stress can provoke episodes of RAS, but the association is not invariable.

There is no indication that aphthous ulcers are related to menstruation, pregnancy and menopause.

There are a few patients whose RAS remits with oral contraceptives or during pregnancy.

Smokers appear to be affected less often.

There often is a genetic basis for RAS. More than 42% of patients with RAS have first-degree relatives with RAS. The likelihood of RAS is 90% when both parents are affected, but only 20% when neither parent has RAS. It also is likely to be more severe and to start at an earlier age in patients with a positive family history than in those without.

Diagnosis

The diagnosis of RAS is made on the basis of history and clinical criteria, since there are no specific laboratory tests available. A medical history should be taken to rule out other ulcerative disorders and conditions such as Crohn’s disease, celiac disease, neutropenia, HIV infection and Behcet’s syndrome.

Differentiation of causes of oral ulceration. RAS: Recurrent aphthous stomatitis. PFAPA: Periodic fever, aphthae, pharyngitis and adenitis syndrome.

Erythema exsudativum multiforme (EEM), Erythema multiforme (EM)

Erythema multiforme (EM) was initially described in 1866 by Ferdinand von Hebra as an acute self-limited skin disease, symmetrically distributed on the extremities with typical and often recurrent concentric "target" lesions. The term EM minor was proposed later to differentiate the mild cutaneous syndrome from the more severe form, EM major, which involves several mucous membranes.

Stevens-Johnson syndrome (SJS) was considered an extreme variant of EM for many years, while toxic epidermal necrolysis (TEN) was considered a different entity. However, in 1993, agroup

of medical experts proposed a consensus definition and classification of EM, SJS, and TEN based on a photographic atlas and extent of body surface area involvement. According to the consensus definition, SJS was separated from the EM spectrum and added to TEN. Essentially SJS and TEN are considered severity variants of a single entity. The two spectra are now divided into (1) EM consisting of erythema minor and major (EMM) and (2) SJS/TEN. The clinical descriptions are as follows:

EM minor - Typical targets or raised, edematous papules distributed acrally

EM major - Typical targets or raised, edematous papules distributed acrally with involvement of one or more mucous membranes; epidermal detachment involves less than 10% of total body surface area (TBSA).

SJS/TEN - Widespread blisters predominant on the trunk and face, presenting with erythematous or pruritic macules and one or more mucous membrane erosions; epidermal detachment is less than 10% TBSA for SJS and 30% or more for TEN.

Pathophysiology

Pathophysiology of EM is not completely understood but appears to involve a hypersensitivity reaction that can be triggered by a variety of stimuli, particularly bacterial, viral, or chemical products.

A recent international prospective study showed that the major cause of EM is herpes virus. It appeared to play a smaller role in SJS/TEN. In fact, recent or recurrent herpes was the principle risk factor for EMM. Drugs were found to be a more common trigger for SJS/TEN.

Histopathologic characteristics include a lymphocytic infiltrate at the dermal-epidermal junction and around dermal blood vessels, dermal edema, epidermal keratinocyte necrosis, and subepidermal bullae formation. Histology and immunochemistry studies have shown that inflammatory infiltrates of EM and SJS/TEN are strikingly different in density and nature. EM has a high density of cell infiltrate rich in T-lymphocytes. By contrast, SJS/TEN is characterized by a cell-poor infiltrate of macrophages and dendrocytes with strong TNF-alpha immunoreactivity. Immune complex deposition is variable and nonspecific. In severe cases, fibrinoid necrosis can occur in the stomach, spleen, trachea, and bronchi.

Physical

- Symmetrically distributed, erythematous, expanding macules or papules evolve into classic iris or target lesions, with bright red borders and central petechiae, vesicles, or purpura.
- Lesions may coalesce and become generalized.
- Vesiculobullous lesions develop within preexisting macules, papules, or wheals.
- Rash favors palms and soles, dorsum of the hands, and extensor surfaces of extremities and face.
- Postinflammatory hyperpigmentation or hypopigmentation may occur.
- Eye involvement occurs in 10% of EM cases, mostly bilateral purulent conjunctivitis with increased lacrimation.
- Mucous membrane blistering occurs in about 25% of cases of EM, is usually mild, and typically involves the oral cavity.

Eruptions occur symmetrically on the extensor aspects of the joints (e.g., the dorsal hands, elbows, knees) as erythematous papules or edematous erythema, and they spread centrifugally in about 48 hours to form sharply circumscribed, round or irregularly shaped erythema. The center of the erythema is concave, presenting either as a target lesion or iris formation, also called exudative erythema. The affected area simultaneously shows new and old lesions that may fuse into map-like patterns. EM may be accompanied by blistering (bullous EM) and erosions of the oral mucosa. EM frequently occurs in the young and middle-aged, and it tends to appear during the spring and

summer. Infectious symptoms including high fever and pharyngodynia may precede the onset. In cases caused by herpes simplex infection, EM tends to occur 1 to 3 weeks after the onset of the herpes simplex symptoms (post-herpetic EM).

Pathogenesis

EM is caused by various factors, such as viral or bacterial infections (by herpes simplex or Mycoplasma pneumoniae), and drugs and malignant tumors. It is estimated that EM is a cell-mediated immuno reaction leading to the destruction of keratinocytes expressing various antigens. However, the underlying pathomechanism is not known.

Pathology

In the early stages of epidermal EM, there is lymphocytic infiltration into the dermo-epidermal junction and vacuolar degeneration of basal cells. As the disease progresses, lymphocytes (CD8+T cells) infiltrate into the epidermis and necrosis of epidermal cells and subepidermal blistering are found.

Laboratory findings

Because of inflammation, CRP may be positive and the erythrocyte sedimentation rate is elevated. The herpes simplex virus antibody titer, Mycoplasma antibody titer and antistreptolysin O (ASO) titer may be elevated in some cases. In cases involving bacterial infection, there is an increase in neutrophils.

Diagnosis, Differential diagnosis

EM is relatively easy to diagnose by its characteristic clinical features and by the distribution of the eruptions. History of previous diseases, such as infectious diseases, supports the diagnosis.

Urticaria - Itching is more severe. Each lesion usually disappears within 24 hours. Dermographism rubrum occurs.

SLE - Systemic symptoms occur (renal, arthritic, etc.). Laboratory findings of antinuclear antibodies, etc. Erythema multiforme sometimes occurs in association with SLE.

Bullous pemphigoid - Direct/indirect immunofluorescence reveals antibodies against basement membrane.

Comprehension control

1. Main and additional methods of diagnosis of the patients with Acute herpetic stomatitis and chronic herpetic stomatitis.
2. Differential diagnosis of the Acute herpetic stomatitis and chronic herpetic stomatitis.
3. Main and additional methods of diagnosis of the patients with fungal diseases in children.
4. Differential diagnosis of the fungal diseases of the oral mucosa in children.
5. Differential diagnosis of the cheilitis in children.
6. Immediate and delayed types of allergic reactions.
7. Anaphylactic shok.
8. Quincke's edema.
9. Urticaria.
10. Differential diagnosis of the immediate types of allergic reactions.
11. Diagnosis and differential diagnosis of the medicamentous allergy on the oral mucosa.
12. Diagnosis and differential diagnosis of Exudative erythema multiforme.
13. Diagnosis and differential diagnosis of Steven-Jonson syndrome.
14. Acute mechanical traumas of the oral mucosa in children.
15. Bednar's aphthae.
16. Decubital ulcer.

17. Thermal, chemical and radiation trauma.

Test control

1. In a minute after torus anaesthesia was introduced with 2% solution of novocaine of 4 ml on the occasion of the 17 tooth extraction, a patient complained of a difficult breathing. Objectively: upper and lower lips, mucous membrane of larynx and oral cavity are swelled and hyperemic. What complication occured in the patient?

A. Quincke's edema
B. Intoxication by anaesthtetics
C. Anaphylactic shock
D. Collapse
E. Coma

2. According to the mother, a 5-year-old child complains about pain during swallowing, weakness, body temperature rise upt to 39,5oC, swelling of submental lymph nodes. Objectively: the child's condition is grave, body temperature is 38,8oC. Mucous membrane of the oral cavity is markedly hyperaemic and edematic with haemorrhages and ulcerations. Pharynx is markedly hyperemic, lacunae are enlarged and have necrosis areas. Regional, cervical, occipital lymph nodes are painful, enlarged and dense. What is the most likely diagnosis?

A. Infectious mononucleosis
B. Acute herpetic stomatitis
C. Herpetic angina
D. Necrotizing ulcerative gingivostomatitis
E. Lacunar tonsillitis

3. A 10-year old boy complains of swelling of the lower lip, which appeared suddenly after wasp sting and difficult breathing. Objectively: lower lip is in three times bigger than normal, the skin in the area of edema is pale. There is swelling of the tongue in the mouth. Oral mucosa of the soft palate is swollen. What drugs should be used firstly?

A. Antihistamines
B. Corticosteroids
C. Anti-inflammatory drugs
D. Antibacterials
E. Analgesics

4. A 4,5-year-old child presents with eruptions on skin and in the mouth which appeared on the previous day. Objectively: the child is in medium severe condition, body temperature is 38,3oC. Scalp, trunk skin and extremities are covered with multiple vesicles with transparent content. Mucous membrane of cheeks, tongue, hard and soft palate exhibits roundish erosion covered with fibrinous film. Gums remain unchanged. Submandibular lymph nodes are slightly enlarged. What diagnosis can be assumed?

A. Chicken pox-induced stomatitis
B. Acute herpetic stomatitis
C. Exudative erythema multiforme
D. Measles-induced stomatitis
E. Scarlet fever-induced stomatitis

5. A 9-year-old child complains of increase of body temperature to 38,5 ° C, sore throat, weakness. There is an acute catarrhal stomatitis in the mouth . Tonsils are swollen, hyperemic, coated with yellow-gray coating that is easy to removed. Submandibular, cervical, occipital lymph nodes are significantly enlarged, slightly painful to palpation. It was revealed leukocytosis and atypical mononuclear cells in blood. Define the causative agent.

A. Epstein-Barr virus
B. Herpes simplex virus
C. Streptococcus haemolytica
D. Coxsackie virus
E. Herpes simplex virus

6. A 10 year-old child has been complaining of pain in the throat, cough, increase of temperature to 38 C for 2 days. An objective examination revealed an acute catarrhal stomatitis. Tonsils are swollen, hyperemic, coated with yellow-gray coating. The coating is crumbly and easy to remove. Submandibular, cervical lymph nodes are significantly enlarged, painful to palpation. The laboratory study found leucocytosis and monocytosis . Define the most likely diagnosis.

A. Infectious mononucleosis
B. Varicella
C. Measles
D. Scarlet fever
E. Diphtheria

7. A dry cough, runny nose, increase of the temperature to 38,3° C is observed in a 3-year-old child. Objectively: there are white and gray pin-point spots, surrounded by a bright red rim on the hyperemic oral mucosa adjacent to molars. There are pink enanthema on the soft palate. What is the most likely diagnosis?

A. Measles
B. Diphtheria
C. Scarlet fever
D. Chickenpox
E. Herpetic angina

8. Parents of a 6- year-old girl appealed to the dentist for a consultation . Child is ill since yesterday with increasing of the body temperature to 38.3 C, runny nose and a cough. Objectively: there are pin-pointed whitish-gray spots, surrounded by a bright red rim on the swollen, hyperemic buccal mucosa adjecent to molars. There are the bright red spots of irregular shape on the mucous membrane of the soft palate. What is the primary clinical manifestation of the disease?

A. Belsky-Filatov-Koplik's spots
B. Burning pharynx "
C. Erosive element
D. "Strawberry tongue"
E. Polymorphism of primary cells

9. A 16-year-old patient complains of pain during swallowing, increase of the temperature to 38.0 C, which arose day ago. Objectively: there are white coating with clear borders on the mucosa of the tonsils. This coating is tightly attached to the adjacent tissues and is difficult to remove.

Regional lymph nodes are enlarged , painful. There is edema of the subcutaneous adipose tissue. What is the probable diagnosis ?

A. Diphtheria
B. Acute candidiasis
C. Infectious mononucleosis
D. Chickenpox
E. Sore throat

10. After physical examination it was revealed a whitish-gray coating on the tonsils and throat mucosa. Dentist preliminarily diagnosed diphtheria. The doctor refered the patient for the bacteriological examination. Which pathogen will confirm the diagnosis?

A. Loeffler bacillus
B. Candida Fungi
C. Pale treponema
D. Streptococcus haemolytica
E.

Recommended literature

1. Paediatric dentistry/ Richard Welbury, Monty Duggal – 3rd ed., 2005 Copyright.
2. L.A. Khomenko. Pediatric Therapeutic Dentistry.- K.:Book-plus, 2012

PRACTICAL CLASS № 9

Theme: Main principles of treatment of diseases of oral mucosa in children (viral, bacterial, traumatic, allergic origin and during somatic diseases).

Objective: To learn with students the main principles and methods of treatment of diseases of the oral mucosa in children.

Pre-study test questions

1. Primary and secondary lesions of the oral mucosa.
2. Classifications of the diseases of the oral mucosa.
3. Traumatic lesions of the oral mucosa.
4. Viral diseases of the oral mucosa in children (acute and chronic herpetic stomatitis).
5. Peculiarities of the clinical course of an acute viral and infectious diseases in children (measles,

chicken pox, shingles, scarlet fever, diphtheria, whooping cough, infectious mononucleosis, AIDS).

6. Fungal diseases in oral cavity.
7. Manifestations of allergic diseases in oral cavity.
8. Cheilitis and glossitis.
9. Manifestations of endocrine diseases, diseases of the gastrointestinal tract blood diseases.
10. The main groups of the medicines and its pharmacodynamics.

Contents of the class

Traumatic/Irritational Lesions Differential diagnosis of white lesions depends on whether the lesion can be rubbed off when wiped with a piece of gauze. A white plaque-like lesion found in an area of irritation may be irritational keratosis due to constant irritation from a dental appliance (e.g., denture, orthodontic wire) or biting. Although these lesions do not rub off, they are not considered malignant. Treatment consists of removing the irritation. If the lesion does not disappear in a few months, the patient should be referred to a dental specialist.

A fibroma usually appears as a submucosal, smooth, pink nodule on the lateral border of the tongue, buccal mucosa, or lower lip where there is chronic irritation from biting or friction. These lesions are asymptomatic and do not have a malignant etiology. Management comprises monitoring and surgical excision.

A mucocele develops when a minor salivary gland duct in the inner part of the lip (usually lower lip) is severed and the secretions spill into the tissues. Clinically, it appears as a swelling that is bluish or the normal pink color of the mucosa. Its size may increase or decrease over time. These lesions should be monitored and can be surgically excised.

Candidiasis

Oral candidiasis can exhibit a variety of clinical patterns and is predominately caused by an overgrowth of the *Candida* species, the most common being *Candida albicans*, which is part of the normal flora.

Incidence varies by age and certain predisposing factors, such as HIV infection, smoking, dentures, chemotherapy, diabetes, corticosteroid therapy, xerostomia (dry mouth), and short- or chronic-term broad-spectrum antibiotic use.

Acute pseudomembranous candidiasis, or thrush, is the most common form of oral candidiasis, presenting as white, creamy, elevated plaques that easily rub off with gauze, leaving a painful, raw, ulcerated surface. The most typical sites include buccal mucosa, dorsal tongue, and palate. Diagnosis is usually based on the clinical appearance, with or without confirmation by smear or culture of *Candida*, because no chairside test (in the office, not sent to the laboratory) exists for oral candidiasis

Topical antifungals (clotrimazole) are the drugs of choice for uncomplicated, localized thrush in patients with normal immune function. Systemic antifungals are usually indicated in cases of disseminated disease and/or in immunocompromised patients. It is important for patients to continue the drug at least two days after lesions resolve. If patients use a corticosteroid inhaler, they should be instructed to brush their teeth and rinse their mouth after every use. Acidophilus or yogurt is recommended to take with broad-spectrum antibiotics to reduce the incidence of candidiasis infection.

Chronic atrophic (erythematous) candidiasis, or denture sore mouth, appears as a red patch or velvet-textured plaque on the hard palate under a denture. The most common causes are poor denture hygiene and continuous wear of the denture.The patient should be instructed to clean the denture on a daily basis and not to wear it while sleeping.Topical antifungal creams or ointments can be applied to the inner lining of the denture. Additionally, nystatin pastilles or clotrimazole troches may be used.

Angular cheilitis, another form of candidiasis, ranges from slight erythema, with superficial scaling fissures at the corners of the mouth, to intensely red and ulcerated lesions, accompanied by soreness and a burning sensation. The etiology is multifactorial and includes *C. albicans* and *Staphylococcusaureus* infection, as well as a deficiency of vitamin B and folic acid.[9] Topical antifungal creams and ointments are the treatment of choice.

Leukoplakia and Erythroplakia

Since the oral cavity is composed of many different types of tissues, benign or malignant neoplasms may occur. According to the World Health Organization, leukoplakia is defined as "a white patch or plaque that cannot be characterized clinically or pathologically as any other disease." Erythroplakia has the same definition except that it is a red patch. Speckled leukoplakia is a combination of red and white lesions.

The primary risk factor for oral cancer is tobacco use.[11] Heavy alcohol consumption (more than four drinks per day) is a higher synergistic risk factor when combined with heavy tobacco use.[12] Pharmacists should immediately refer patients to an oral surgeon or otolaryngologist if they have any red or white patches in the mouth.

Lichen Planus

Lichen planus is a common dermatosis that occurs on the skin and oral mucosa. About 50% of patients who have oral lesions also present with skin lesions.[13]The tongue and buccal mucosa are the most frequent sites.

Among the different forms of lichen planus, the reticular form is the most common and appears as slightly elevated, fine, white lines called *Wickham's striae*, which have a lace-like pattern. The etiology of lichen planus is not clear; however, it does involve an immunologically induced degeneration of the superficial epithelium.

Treatment for lichen planus, such as topical corticosteroids (e.g., fluocinonide) and/or corticosteroid mouthwashes, is usually initiated if lesions are symptomatic.

Ulcerations

Recurrent minor aphthous stomatitis, typically referred to as canker sores, is the most common recurrent lesion in the mouth, with a higher incidence in females. Although the etiology of minor aphthous ulcers is essentially unknown, hypersensitivity to streptococcal antigens, stress, and hormonal changes have been proposed.Aphthous stomatitis is not a viral infection, and it is not infectious; however, a genetic predisposition maybe present. Additionally, patients should be evaluated for vitamin B_{12} or folic acid deficiency.

Clinically, aphthous ulcers first appear as a papule, not a vesicle, on movable mucosa only (e.g., inner lower lip, lateral border of the tongue, soft palate). The ulcerations are usually round with a depressed center and a red margin. If there are several, they do not coalesce. They may be painful, especially when speaking and eating. The lesions usually heal in about seven to 10 days, but recurrence is common. It is rare to have lymphadenopathy or fever associated with these lesions.

In most cases, the natural history of aphthous ulcers is one of eventual remission. Treatment is usually palliative and supportive in patients with significant discomfort. Relief of pain and reduction of ulcer duration are the main goals of therapy. Medications include topical corticosteroids, analgesics, and antimicrobials.

Topical corticosteroids are the mainstays of treatment but only reduce the pain, not the rate of recurrence. Topical analgesics, including benzocaine and viscous lidocaine 2%, provide pain relief. Amlexanox (Aphthasol) 5% paste, an inhibitor of the formation and release of inflammatory mediators from mast cells and neutrophils, is applied no more than four times daily and may increase healing and decrease pain but does not reduce the frequency of recurrent ulcer episodes.

Limited randomized, controlled studies support the use of tetracycline. One 250-mg capsule was dissolved in 180 mL of water, and the patient was instructed to rinse four times per day for about five days. Another option is to rinse with 5 mL of tetracycline syrup 250 mg/5 mL four times daily for five days.

Chlorhexidine gluconate, an antimicrobial oral rinse, may reduce the severity and pain but not the frequency of recurrence. In addition, immune modulators such as thalidomide are used to treat recurrent aphthous ulcers in HIV-infected patients. These ulcers often have protracted healing times.

Alternative therapies without scientific evidence include zinc lozenges (one lozenge four to six times daily), vitamin C (500 mg four times daily), and vitamin B complex (one tablet four times daily).

Vesicular Lesions

Oral Herpes Simplex Virus Infection: Herpes simplex virus (HSV), a DNA virus, is usually classified by anatomical location. HSV-1 infections, termed herpes labialis, cold sores, or fever blisters, are located above the torso and are typically associated with oral lesions. HSV-2 infections, termed herpes genitalia, are located below the torso and are usually linked to genital lesions. However, in about 30% of cases, HSV-1 can cause genital herpes by oral-genital or genital-genital contact with a person infected with HSV-1.

Primary Herpetic Gingivostomatitis: The initial HSV-1 outbreak usually occurs in childhood when the virus is transmitted through such activities as kissing an infected person or drinking from the same cup of a person with open lesions. The peak incidence of primary HSV-1 is predominately between two to three years of age, although adults can also be diagnosed with this disease. The patient usually presents with fever, headache, lymphadenopathy, malaise, sore throat, and nausea and vomiting. Later, small vesicles appear in the mouth; they eventually rupture, leaving small painful ulcers covered by yellow-gray membranes.[19] In addition, there is generalized acute gingivitis.

Treatment of primary acute herpetic gingivostomatitis in healthy children and adults, including fluids and acetaminophen, remains palliative. Early treatment with acyclovir may shorten the duration of all clinical manifestations and infectivity of affected children. Antibiotics are used only to prevent secondary infection.

Recurrent Intraoral Herpes/Herpes Labialis: After primary exposure, this virus remains dormant in the trigeminal ganglion until a later date when it is reactivated. Recurrences are thought to be due to stress, sunlight, fever, HIV infection, certain foods, menstruation, trauma (e.g., after a dental procedure), or other irritants.[21]The primary infection is more severe than recurrent infections. Although not everyone develops recurrent infections, some may have many recurrences per year. Prodromal symptoms of mild tingling, burning, or itching may occur during the 12 to 36 hours preceding vesicle eruption. The vesicles develop into ulcers, which finally crust and then heal within 24 to 48 hours. Spontaneous healing occurs over seven to 10 days. Ulcers are found primarily on nonmovable mucosa (e.g., lateral border of the tongue and gingival, perioral, and vermillion border of the lips). These lesions are referred to as herpes labialis, cold sores, or fever blisters.

Treatment consists of controlling symptoms and shortening the duration of active infection. Lesions are usually mild and self-limited. Topical antiviral and analgesic agents can help decrease healing time and pain. Acyclovir 5% is an accepted standard topical therapy. Systemic antiviral agents, such as acyclovir, valacyclovir, and famciclovir, may reduce the duration of symptoms but are helpful only for prodromal symptoms.[23] These agents are not curative, but they may lessen the severity and duration of infection and rate of recurrence. Since this is a viral infection, antibiotics should not be used. Corticosteroids have not been effective in treating herpes.

Docosanol (Abreva) 10% topical cream inhibits fusion of the virus with the human cell membrane, blocking entry and subsequent viral replication. It is designed to speed healing time of

recurrent oral/facial herpes, and it stopped progression to the blister stage in about 34% of patients who apply it at the early sign of an outbreak (redness). This OTC medication should be applied five times per day until the infection is healed.

Xerostomia: Xerostomia can be due to medications (e.g., antipsychotics, anticholinergics, antihistamines, diuretics), illness (e.g., HIV or cancer), or aging. Salivary glands receive sympathetic and cholinergic innervation. Activation of either autonomic system will increase secretions, but the cholinergic activation is the more important system. Anticholinergic drugs bind to the cholinergic (muscarinic) receptors, thus inhibiting salivary secretions from the salivary glands, resulting in dry mouth.

Treatment is aimed at increasing secretions or moisturizing the mouth. Saliva substitutes, or artificial saliva, have the necessary ions, buffering properties, and lubricating mucins. OTC products include Oasis, Biotène, Optimoist, Xero-Lube, and Salivart. Dentists or physicians may prescribe cholinergic drugs, such as Salagen (pilocarpine) and Evoxac (cevimeline).

Comprehension control

1. General principles of treatment of diseases of the oral cavity.
2. Name the main stages of the local treatment of the catarrhal gingivitis.
3. Name the main stages of the local treatment of the erosive lesions.
4. Main principles of treatment of the traumatic lesions of the oral mucosa.
5. Medicines for antiseptic processing of the oral cavity.
6. Medicines for anaesthetic processing of the oral cavity.
7. Antibiotics for treatment of the diseases of the oral mucosa.
8. Anti Inflammatory drugs for treatment of the diseases of the oral mucosa.
9. Antiviral drugs for treatment of the diseases of the oral mucosa.
10. Antihistamine drugs for treatment of the diseases of the oral mucosa.
11. Medicines for stimulation of the epithelization of the lesions.

Test control

1. During dental examination of 4-years-old patient was diagnosed with acute herpetic stomatitis (mild form). Which medicine should be applied for the aethiological therapy?

A. antiviral medication
B. anti-bacterial medication
C. pain control
D. stimulation of epitalisation
E. hygienic education

2. During dental examination of 2- years-old patient diagnosis of acute herpetic stomatitis (mild form) was diagnosed. Which medicine should be applied for the stimulation of epitalisation?

A. tocopherol acetate
B. antiviral medication
C. anti-bacterial medication
D. trypsin
E. glucose solution

3. During dental examination of 3- years-old patient diagnosis of acute herpetic stomatitis (severe form) was diagnosed. Which medicine should be applied for the desintoxication?

A. intravenous injections of 10 % glucose solution
B. tocopherol acetate
C. antiviral medication
D. anti-bacterial medication
E. trypsin

4. During dental examination of 11-years -old patient acute herpetic stomatitis was revealed. Which medicines of anti-viral action should be applied for the local treatment?
A. Acyclovir (zovirax)
B. Solcoseryl
C. Trypsyn
D. Deoxyribonuclease
E. All mention above

5. A 10-year o;d patient appealed with complaints of pain during eating. Objectively: there are hyperemic areas on mucosa of the hard palate and lower lip. Erosion which is covered with fibrinous film is observed on the lip. The appearance of these complaints relates to receiving sulfonamides. Diagnosis: toxic-allergic stomatitis. What treatment will you choose?
A. Antihistamines
B. Antiseptics, keratoplasty
C. Antibiotics, sulfonamides
D. Antiviral, immunomodulators
E. Corticosteroids

6. An 1 year old child is restless, refuses to eat, the body temperature is 37.7 C. Objectively: mucosa of cheeks, vestibulum oris, and tongue is hyperaemic and covered with cheesy white coating. What medications should be prescribed firstly?
A. Antifungal
B. Antibiotics
C. Antiviral
D. Antiallergic
E. Antiseptic

7. The patient of 14 years old is being treated in hospital with exacerbation of chronic colitis. Objectively: there are four round-shaped erosions in the area on the lateral surface of flushed tongue and in the area of transitional fold near by tooth 45. What drugs should be used for the treatment of elements during first visit of the dentist?
A. Anesthetics, antiseptics
B. Painkillers, antiviral drugs
C. Painkillers, antifungal drugs
D. Painkillers , antibiotics
E. Keratoplastyc substances

8. The 15 years old child complains of the presence of several painful erosions in the mouth cavity. The erosions appear and disappear during several days, and in 3-4 months period they appear again. Objectively: there are round-shaped erosive elements on the mucosa of lower lip sized

6.5 mm with sharp edges, surrounded by flushing rim, covered with a grayish coating, sharply painful on palpation. After the examination, diagnosis was clarified: HRAS. What drugs for topical treatment should be applied to reliase swelling and inflammation in the early stage of treatment?

A. Corticosteroids
B. Anesthetics
C. Keratoplastic substances
D. Antimicrobial drugs
E. Hyposensibization medicines

9. A child of 11 years old complains of presence of painful lesion in the mouth caviry which makes eating difficult. Similar symptoms were observed for the first time 2 years ago. There is an painful small-sized oval erosion, covered with grayish-white color on the bottom of the mouth. The erosion is hyperemic and has infiltrated edges. What local treatment scheme should be chosen for treatment of this pathology?

A. Anesthesia, antiseptics, keratoplastics
B. Anesthesia, causal therapy, keratoplastics
C. Removal of traumatic factor, suturing damage
D. Elimination of irritating factor, antiseptics, analgesics, keratoplastics
E. Hyposensetization, keratoplastics

10. A 12-year-old boy complains of fever up to 38C, weakness, headache, pain in the mouth, presence of vesicles and ulcers. The acute condition developed three days ago. The patient has a history of recent pneumonia treated with antibiotics. Objectively: oral mucosa is hyperemic and edematous. The mucosa of lips, tongue and cheeks has large erosions covered with fibrinous pellicle. The lips are covered with thick brown crusts. The back of the hand has papules of double-contour color. Which of the listed agents should be primarily used in the topical treatment?

A. Painkillers
B. Antiinflammatory
C. Antiviral
D. Antimicrobial
E. Antifungal

Recommended literature

1. Paediatric dentistry/ Richard Welbury, Monty Duggal – 3rd ed., 2005 Copyright.
2. L.A. Khomenko. Pediatric Therapeutic Dentistry.- K.:Book-plus, 2012