Poltava State Medical University
Chair of Propaedeutics of therapeutic stomatology

NOT-CARIOUS DEFECTS OF TEETH

Lecture for III-year students
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Lecture plan

- Classification of not-carious defects of teeth;
- 2. Enamel hypoplasia: etiology, types, clinic, treatment;
- 3. Enamel hyperplasia: types, clinic, treatment;
- 4. Fluorosis: etiology, clissification, clinic, treatment;
- 5. Hyperesthesia: types, clinic, treatment;
- Wedge-shapped defect: theories of development, clinic;
- 7. Erosion of hard tooth tissues: clinic;
- 8. Necrosis of hard tooth tissues: types, stages, clinic;
- 9. Tooth traumas: types, clinic, treatment.

Classification of not-carious defects of teeth by M. I. Groshikov (1985)

I. Lesions, arising during teeth development:

Hypoplasia of enamel;

Hyperplasia of enamel;

Endemic fluorosis of teeth;

Anomalies of development and teething;

Hereditary infringements of development of teeth.

II. Lesions, arising after the dentition:

Teeth pigmentation and dental deposit;

Abrasion of hard tissue of teeth;

Wedge-shaped defect (clinoid);

Erosion of teeth;

Necrosis hard tissue of teeth;

Traumas of teeth;

Teeth hyperesthesia.

Enamel hypoplasia –

defect of enamel development, which is characterized its insufficient forming and mineralization and shows up as spots or defects on the surfaces of enamel, which have irreversible current and remain on teeth for life.

Etiology. Hypoplasia of tooth tissue (usually enamel) occurs because of metabolic processes in the follicle of tooth under the influence of malfunction of the mineral and albumineus metabolism of fetus or child.

Temporary teeth hypoplasia, which are forming during the intrauterine period, caused by the metabolic imbalance of the pregnant (German measles, toxoplasmosis, toxicosis, rhesus incapability). More often arised in premature children.

Permanent teeth hypoplasia occurs under the influence of different somatic diseases (rachitis, tetany, acute infectious diseases, gastrointestinal diseases, toxic dyspepsia, nutritional dystrophy, cerebral malfunction), which occur during formation and mineralization of these teeth (period from 6 month to 1 - 1,5 year).

Types of hypoplasia

SYSTEM HYPOPLASIA - characterized by the defeat of all teeth, or group of teeth, which is formed in same interval of time. More frequent develops for children in the first 9 months after birth. It is arisen up on general reasons:

- Indicate of antibiotics for the children of first-year of life, which repress the function of enameloblasts (tetracycline);
- Illnesses of the endocrine system;
- Illnesses of cns;
- Rachitis;
- Acute infectious diseases;
- Heavy form of dyspepsia.

Types of hypoplasia

1. Systemic hypoplasia





2. Local hypoplasia



Forms of system hypoplasia Change of enamel color









Change of color- weak degree of enamel hypoplasia, which is shown in the view of simmetric spots often white, less yellowish color, with a clear border and identical size on the groups of teeth with the same name. Spots at hypoplasia aren't painted by dyes (unlike initial caries(white spot). Disease has painless current.

Forms of system hypoplasia Enamel underdevelopment

wavy enamel dotty enamel furrowed enamel













Forms of system hypoplasia Enamel absence (aplasia)



The most seldom form. It is characterized by lack of enamel on a certain site of tooth. At this form there can be complaints of painful feelings from thermal and chemical irritants.

Veriety of a systemic hypoplasia

Getchinson's teeth

Maxilla central incisors have a barrel-like form of a crown or a screw-driver form (the size at a neck more than in cutting edges) and semi-lunar cutting at cutting edge.

Pflyuger's teeth

anomaly of the first molars at which the size of a crown at a tooth neck more than at cutting edge, and chewing hillocks are underdeveloped. Reason of development: inborn syphilis

Fournier's teeth

the central incisors are similar to Getchinson's teeth (tubby form), but without semi-lunar cutting. Reason of development: inborn syphilis











Local hypoplasia

The cause of the local hypoplasia is a trauma of the follicle of the permanent teeth. Defects can occur because of jaw fracture, temporary tooth dislocation, inflammatory processes (periodontitis, periostitis, osteomyelitis). Trauma or intoxication cause malfunction of ameloblast, sometimes odontoblast, there for tooth take the wrong shape and has unusual form. More often the permanent premolars are damaged because their follicle are situated between roots of temporary molars, which very oftenare affected by periodontitis.











Treatment of hypoplasia

LOCAL:

- Bleaching of spots (peroxide carbamide) and enamel remineralization;
- Grinding of spots and coverage those areas by replacing substances which contain minerals (preparations with Ca, fluorine (75 % paste of fluoride Na (by Lukomsky), 0,4-1-2 % solution s of fluoride Na, " Фторлак" (varnishes) (5 % fluoride Na, 40 % fir balm, 10 % shellac, 12 % chloroform, 24 % ethyl spirit), «Белак F», "Fluor Protector", "Duraphat", "Bifluorid 12", «Белагель Са/Р", «Белагель F»);
- Restoration of the tooth anatomic form by composite materials.
- Restoration of the tooth anatomic form by orthopaedic crowns.

COMMON:

- Reception of Calcium containing remedies per os;
- Reception of antioxidants and microelementss;
- Reception of multi vitamin preparations
- Electrophoresis 10 % sol. gluconate Ca, Calcium chloride, lactate Ca,
 2,5 % sol. glycerophosphate Ca,
 5-10 % sol. phosphate calcium 3 times on a year.

Enamel hyperplasia (ENAMEL PEARLS, DROPS)

Surplus formation of hard tooth tissues takes place during his development.

Localized on the contact surfaces of premolars and molars in area of neck of tooth, bifurcation of teeth roots on enamel – cement border. Does not

show up clinically.



Fluorosis -

is the variety of system hypoplasia which develops as a result of violations of mineral exchange and arises up at the surplus entering fluorine in organism during teeth formation and mineralization.

Prevalence of fluorosis among a population and weight of defeat depends on the concentration of fluorine in a drinking-water, soil, food.

Optimal contentration of fluorine in a drinking-water is 0,8-1,2 mg/l, a max. possible concentration of fluorine in a drinking-water is 1,5 mg/l.

Fluorosis affect teeth of children who developed fetally, and then resided in the endemic centers in period of formation of enamel. Therefore this disease doesn't arise at the adults living in the focus of an endemic fluorosis.

Classification of fluorosis by V.K. Patrikeev

1 group - displasia - teeth defeat without loss

of tooth tissues

- the dashed;
- the spotty;
- the chalked-dotty.
- 2 group hypoplasia teeth defeat with the

loss of tooth tissues

- the chalked-dotty
- erosive;
- the destructive.



Classification of fluorosis by A.K. Nikolishin (1989)

on the basis of which lies the definition of fluorescence in UV-rays, indexes of ohmic electrical resistance of hard tooth tissues

- I. Light degree (without slaking of primary fluorescence of enamel). An enamel contains hydroxifluorapatite, stable compound which does not change the clinical displays during life.
- II. Middle degree (poorly expressed fragmentary slaking of fluorescence of enamel; without destruction). Accompanied by fragmentary formation of calcium fluorine. Teeth cut mostly uncolouring, and than in accordance with mineralization of enamel after some time the fragmentary colouring of enamel originates from light to dark broun color.
- III. Heavy degree (well expressed total slaking of primary fluorescence of enamel). Is accompanied by formation of the well expressed layer of calcium fluorine. Such enamel sharply differs from an enamel with high maintenance of hydroxiapatite.

Fluorosis















Treatment of fluorosis

- The easy degree of fluorosis without cosmetic defeats of enamel does not require of treatment.
- The dashed, spotted, chalked-dotty forms of fluorosis without destruction of enamel treat, applying remineralized therapy during 6 months during 2 years.
- Spotted form of fluorosis on condition of localization of spots on the vestibular surfaces of frontal teeth and with intensive discoloration of enamel treat with method of bleaching with further remineralized therapy.
- At presence of pigmentation, destructions of enamel is performed at first rem. therapy, then bleaching and polish of teeth surfaces (A.K. Nikolishin, 1992).
- Erosive and destructive form of fluorosis restoration of the tooth anatomic form by composite material.
- At considerable destruction of teeth crowns restoration of the tooth anatomic form by orthopaedic crowns.

Hyperesthesia –

Increased sensitiveness of teeth to the action of different irritants - temperature, chemical, mechanical.

Pain arises up as a result of baring of dentine and contact of its nervous receptors with an external environment at:

- baring of teeth necks;
- baring of cement of tooth root;
- pathological abrasion of enamel, dentine;
- erosions of enamel;
- clinoid defect;
- trauma;
- violation of mineralization processes.

Types of hyperesthesia

 LOCAL - it is arisen up from the action of local irritants;

• SYSTEM (general) - it is arisen up at neuropsychic, endocrine, infectious diseases, at a metabolic disturbance, at the disease of gastrointestinal tract.

Classification of hyperesthesia (by Fedorov Yu. A., 1981)

- I. By prevalence:
 - local form;
 - general form.

II. By origin:

- hyperesthesia of dentine which is connected with loss of hard tooth tissues;
- hyperesthesia of dentine, unconnected with loss of HTT III. At clinical current:
 - 1 degree teeth react at cold and warm, EOD 5-8 mkA;
 - 2 degree teeth react at temperature and chemical (salt, sweet, sour, bitter) irritants, EOD 3-5 mkA;
 - 3 degree a tooth react at all types of irritants (included mechanical), EOD 1,5-3,5 mkA.

Hyperesthesia treatment

- 1. Cauterizing (destruction of organic substance of hard tooth tissues leads to formation of coagulater which hinders to moving of dentinal liquid diminishing of the pain feeling) 30% sol. silver nitrate, 10% sol. Zinc chloride, in modern desensitizers-glutaraldegidum.
- **2. Remedies of dehydrating action** alkalis (hydrocarbonate, sodium carbonate, sodium chloride, Magnesium chloride)
- 3. Remedies of biological action (predetermine alteration of biological structure of hard tooth tissues:
- Fluorine containing 75 % paste of fluoride Na (by Lukomsky), 0,4-1-2 % solutions of fluoride Na, "Фторлак" (varnishes), «Белак F», "Fluor Protector", "Duraphat", "Bifluorid 12", «Белагель Са/Р", «Белагель F»; 1-2 % gel of fluoride of sodium on 3 % an agar; 5 % fluoric phosphate-cement; 4 % sol. fluoric tin.
- Calcium containing (10 % sol. gluconate Ca, Calcium chloride, lactate Ca, 2,5 % sol. glycerophosphate Ca, 5-10 % sol. phosphate calcium).
- Deep fluoridation by Knappvost (magnesium-fluoric silicate, then suspension high disperse hydroxide Ca. The complete set: a liquid № 1 typhenfluoride, a liquid № 2 enamel-hermetic liquid)
- 4. Anaesthetic and analgetics (2-5% sol. Dicainum, Lidocainum, Anaesthesinum in glycerin, 4-20% sol. propolis in an alcohol).
- 5. Adhesive systems.

Wedge-shaped defect — (cuneiform defect)

not carious teeth defeat, which is localized in the neck area (more often at vestibularis surface) of all group of teeth.

Etiology. There are a few theories of development:

Mechanical – mechanical damage at using of hard tooth brush, abrasive toothpastes, powders. This theory is confirmed that teeth which come forward from dental row are mostly damaged - canine and premolars and also defeats of teeth on the left in right-hander people and on the right for people which clean teeth by a left hand.

Chemical – damage of teeth under the action of juices citrus.

But a mechanical theory is refuted by that animals

(cows, goats) which not clean teeth have wedge-shaped defects also. A chemical theory is refuted by that differences in an amount wedge-shaped defects between the habitants of south (who use much citrus) and north countries is not found out.



Wedge-shaped defect

Theory of abfracture. In 1984 W.Lee and W.Eakle described the theory of concentration of tension in an enamel as an etiologic factor of wedge-shaped defect. This theory is basis of modern confessedly theory of origin of abfracture. The term of "abfracture" (in translation is a microcrack, microcleavage) was offered J.Grippo in 1991. It is considered, that reason of abfracture are deformations of tooth, arising up because of the excessive occlusal loading. Exactly abfracture is the phenomenon, being the basis of origin of wedge-shaped defects.

Yu. M. Maksimovskiy(1981) an important role in the etiology of notcarious defects of teeth hard tissue takes endocrine violation and, in particular hyperfunctions of thyroid. According to his data, one of symptoms of this disease there is an increase of saliva secretion and decline of its viscidity. It is proved that the patients with thyrotoxicosis have notcaries defects in 2 times more frequent than at healthy.

Clinic of wedge-shaped defect

<u>Complaints:</u> cosmetic defect, inappreciable sensitivity from chemical irritates (acidic, sweet, bitter). Same time complaints are absent, as the pain or sensitivity from irritants does not arise. Defect find out at routine inspection.

Anamnesis of diseases: defects develop very slowly, during a few years. At the beginning hyperesthesia is more expressed, than becomes less.

At survey: at vestibular surface in aria of neck – defect as a wedge, the top of which is directed to the pulp chamber. A defect has two walls – neargam, that located in parallel to the tooth cutting edge (horizontal) and other, which is located under inclined plane and an acute angle is formed. Walls at probing are smooth, dense, withour color change.

Percussion and palpation are painless.

Thermo diagnostic painless or inappreciable short-time painful sensitivity.

EOD: from 2-6 to 20-25 mkA.

Erosion of hard tooth tissues

PROGRESSIVE LOSS OF HARD TOOTH TISSUES (ENAMEL AND DENTINE)

Reasons:

1. Mechanical (tooth brush, tooth-powder); chemical (excessive using of citrus);

2. Disease: gout, endocrine violations (hyperfunction of thyroid gland), neuropsychic diseases, frequent vomiting at bulimia, pregnancy, alcoholism.



Erosion of hard tooth tissues

Stages of development of erosion (E.V. Borovskiy, 1987)

1. Initial – erosion of enamel;

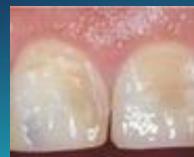


2. Expressed – erosion of dentine.



Stages depending on the depth of defeat (M.Yu.Maksimovskiy, 1981)

I (initial) degree



II (middle)
degree



III (deep)
degree



Erosion of hard tooth tissues

Clinically: symmetric defectson the vestibular surface of incisors, canines, premolars of maxilla, premolars and molar of mandible. Erosion has the appearance of defect of oval or saucer-like form, which is located on the protuberant place of vestibular surface of tooth (equator). Defect has smooth, dense, brilliant surface, probing – sensible, but a probe slides for surface. Erosion is not dyed by methylene dark blue.

Progressive form of not-caries defect, which results to complete losing of tooth and develops under action of unmicrobal factors:

- EXOGENOUS FACTORS: acids, irradiations, radiation
- ENDOGENOUS FACTORS: violation of activity of endocrine glands (hyperthyroidism), disease of CNS, chronic intoxication of organism, inherited violations of odontogeny.

TYPES OF NECROSIS:

- 1. Neargam;
- 2. Acid (chemical);
- 3. Radial;
- 4. Computer. .



Clinically: a whitish spot without accurate borders, the size of which increases gradually, an enamel loses brilliance, becomes mat, rough. An enamel "slides" off from the surface of dentine, very fragile. Hyperesthesia is marked to all types of irritants.



Acid (chemical) necrosis

- ✓ Develops for people which work on the production of inorganic acids (sulphuric, nitric, chlorous), in workshops with the high concentration of fumes of these acids;
- ✓ Develops for patients which have gastritis without secretion of gastric juice and use 10% sol. of chlorous acid.



Computer necrosis

Develops for patients on condition of violations of work rules at the computer (more than 8-10 hours on a day) during 3 - 5 years.

Clinically: localization of defeats on immune areas; in neck areas; defeat has umber color, almost black, the not damaged areas have a turbid greyish color without living brilliance; insignificant hyperesthesia and hyposalivation (xerostomia) is

marked.

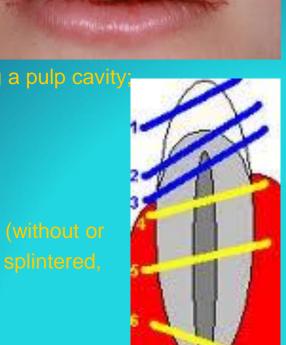
Trauma of teeth

Classification of a mechanical trauma of teeth by Chuprikina N.M. (1985)

- I. Bruise (without or with damage of a neurovascular fascicle).
- II. Dislocation:
 - 1. Incomplete (without damage or with damage of a neurovascular fascicle).
 - 2. Impacted.
 - 3. Complete.

III. Fracture:

- 1. Tooth crown:
 - a) in a zone of enamel;
 - b) in a zone of enamel and a dentine without or with opening a pulp cavity
- 2. Tooth neck:
 - a) is higher than a bottom of a tooth-gum groove;
 - b) below a bottom of a tooth-gum grooves.
- 3. Tooth root:
 - a) with break or without break of a pulp in a place of fracture (without or with shift of fragments): transversal, slanting, longitudinal, splintered, in area cervical, apical and average parts of a tooth root.
- IV. The combined trauma.
- V. Trauma of a dental germ.



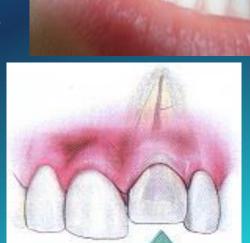
Trauma of teeth

Dislocation

- 1. Incomplete (without damage or with damage of a neurovascular fascicle).
- 2. Impacted.
- 3. Complete.







It is arisen up at lateral or vertical direction of injures force. It can take place with or without the break of vascular-nervous bunch.





Acute trauma

Fracture of tooth crown:

- a) in a zone of enamel and dentine without opening a pulp chamber;
- b) in a zone of enamel and dentine with opening a pulp chamber;















Chronic trauma

Reason of development: harmful habits, professional factors









