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NONCARIOUS TOOTH LESIONS

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МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
КАФЕДРА СТОМАТОЛОГИИ ДЕТСКОГО ВОЗРАСТА

Н. В. Шаковец, Ж. М. Бурак

НЕКАРИОЗНЫЕ ПОРАЖЕНИЯ ЗУБОВ

NONCARIOUS TOOTH LESIONS

Учебно-методическое пособие

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Посвящено проблеме врожденных некариозных поражений зубов. Разобрана схема одонтогенеза и результаты воздействия на него повреждающих факторов на различных этапах. Отражены характерные клинические черты различных нозологических форм некариозной патологии, указаны особенности их рентгенологической картины, рассмотрены диагностика и дифференциальная диагностика. Представлены современные подходы к лечению каждого изолированного вида рассматриваемой патологии. Рассмотрены типы врожденной некариозной патологии как части синдромов и симптомокомплексов, разобрана роль стоматолога в комплексном лечении. Описаны также возможности профилактики некариозных поражений зубов. Первое издание вышло в 2017 г.

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NON-HEREDITARY DEVELOPMENTAL DISTURBANCES OF THE TEETH. ETHIOPATHOGENESIS, DIAGNOSTICS, CLINICAL PICTURE AND TREATMENT

As a group of tooth pathology noncarious defects of the teeth are various in clinical manifestations and their origin. We can divide these defects into: 1) pathology which developed in the period of the follicle development of the tooth (i. e. congenital pathology / before tooth eruption); 2) pathology which developed after tooth eruption. First group in turn can be divided into: 1) non-hereditary developmental disturbances of the teeth; 2) inherited tooth defects.

There are many classifications of congenital noncarious lesions in the world. According to classification of V. K. Patrikeev (1968) the lesions, arising in the period of follicular development, can be divided into:

– enamel hypoplasia;

– enamel hyperplasia;

– endemic dental fluorosis;

– anomalies of the development and eruption of teeth, congenital tooth discoloration;

– inherited tooth malformation.

This clinical classification was common in Belarus and Russia earlier.

Nowadays the most common classification is ICD-D:

K 00 Disorders of tooth development and eruption

K 00.0 Anodontia

K 00.1 Supernumerary teeth

K 00.2 Abnormalities of size and form

K 00.3 Mottled teeth

K 00.4 Disturbances of tooth formation, not elsewhere classified

K 00.5 Hereditary disturbances in tooth structure, not elsewhere classified

K 00.6 Disturbances in tooth eruption

K 00.7 Teething syndrome

K 00.8 Other disorders of tooth development

K 00.9 Unspecified

The most clinically important points (chapters) are the following:

K 00.2 Abnormalities of size and form (enamel hyperplasia)

K 00.3 Mottled teeth (dental fluorosis and non-fluoride enamel opacity)

K 00.4 Disturbances of tooth formation, not elsewhere classified (enamel hypoplasia)

K 00.8 Other disorders of tooth development

Amelogenesis has two stages. At the first stage the organic matrix is formed. At the second stage the matrix undergoes calcification. Interference with normal matrix formation causes defects called hypoplasia. Inteference with calcification and maturation of the enamel produces a condition termed hypocalcification. The different teeth are at their stages of formation in different times (Fig. 1, 2).

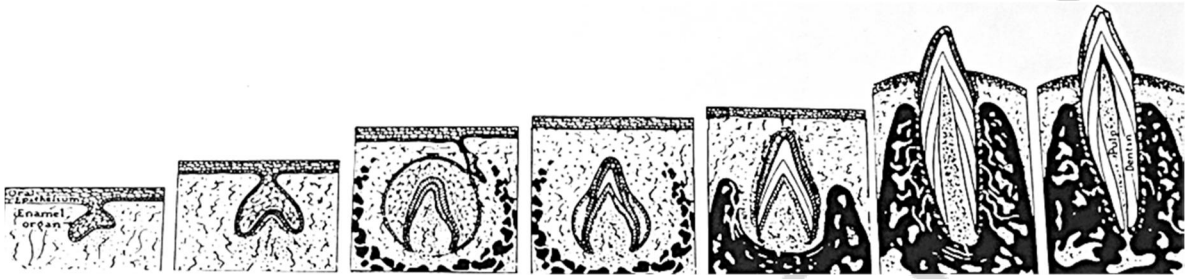


Figure 1. The stages of tooth formation

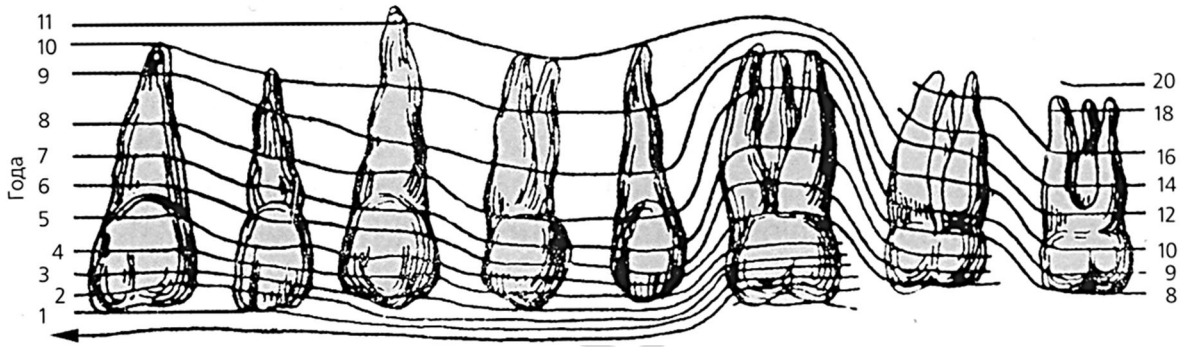


Figure 2. Scheme of mineralization of the permanent teeth

We start the consideration of the clinical types of noncarious lesions from the chapter К 00.4 (Disturbances of tooth formation). It includes the following points:

К 00.40 Enamel hypoplasia
К 00.41 Prenatal enamel hypoplasia
К 00.42 Neonatal enamel hypoplasia
К 00.43 Postnatal enamel hypoplasia
К 00.45 Regional odontodysplasia
К 00.46 Turner tooth
К 00.48 Other specified disturbances in tooth structure, not elsewhere classified
К 00.49 Unspecified

К 00.40 ENAMEL HYPOPLASIA

A hypoplasia of enamel is a teratosis, formed because of violation of metabolic processes in developing teeth and showing up quantitative and quality changes in enamel. Therefore, both stages of amelogenesis are broken.

Histologically we can find decrease of enamel thickness and increase of interprismatic spaces, loss of clearness of enamel prisms and extension of Retzius lines. At electronic-microscopic research of enamel the change of prisms width and orientation of hydroxyapatite crystal is revealed. Enamel hypoplasia is a lesion of group of teeth of one period of mineralization, it is systemic disturbance (Fig. 3). The changes are irreversible (appearing defects remain on the enamel of teeth during all life), violations of structure of dentine and pulp are often marked. The depth of the defect depends on the strength of the stimulus (causal factor), the size — on the duration of stimulus. The number of the defects indicates a multiplicity of stimulus actions Enamel hypoplasia is one of most widely spread noncarious defects, developing in the period of enamel formation.

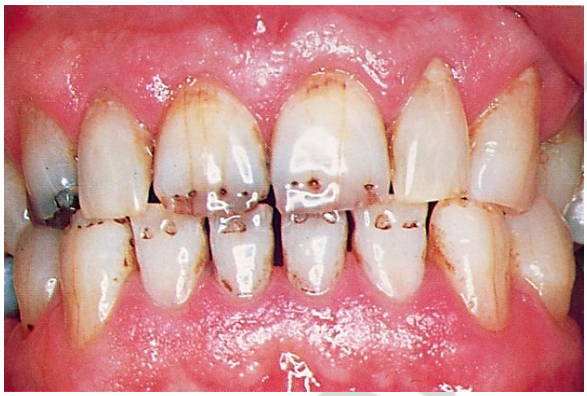


Figure 3. Enamel hypoplasia

The causes of enamel hypoplasia are the following:
– pregnancy pathology;
– diseases of pregnant woman;
– birth defects;
– children diseases;
– malnutrition;
– some medications.

There are some clinical forms of enamel hypoplasia according to the type of changes in enamel:

– *spotted*. The form corresponds with the easy degree of enamel defect and looks like color changes. The symmetrically located spots are visible at one level on masticatory surfaces and cutting edges or on vestibular surfaces in the group of teeth of one period of mineralization (or in all teeth). The enamel of spots is smooth, bright, its color is milk-white, less frequently brown. This form can also be named *Non-fluoride enamel opacity* or *Nonendemic mottling enamel* (K 00.3 Mottled teeth);

– *pit (cup-like)*. The defects are visible as round deepenings which are located horizontally. Pits or cup-like defects aren't connected between themselves, they are more expressed on vestibular and cheek surfaces. A bottom

and walls of the defects are smooth and dense. There is thinning of enamel in the area of defects;

– *furrowed*. The defects are visible as grooves which are located horizontally. A bottom and walls of the defects are smooth and dense;

– *thinning*;

– *aplasia*. It is a heavy degree of enamel hypoplasia, characterized by partial or complete absence of enamel at the part of the crown.

– *combined* (containing previous forms).

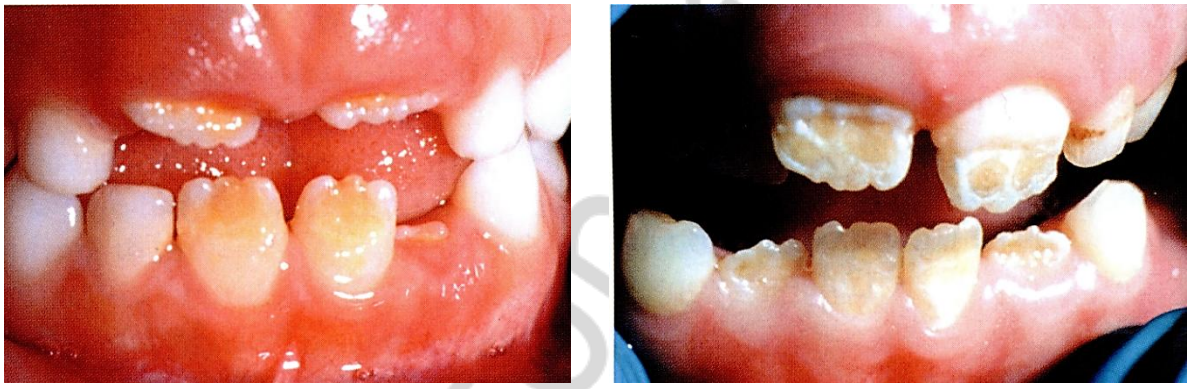
The main clinical features of the enamel hypoplasia are the following:

– teething with already existing defects (Fig. 4);

– the symmetry of the lesions (Fig. 3, 4);

– the uniformity of the clinical manifestations of the same name in the teeth of the same name (for example the central incisors) (Fig. 3, 4);

– the lesions are located parallelly to the cutting (marginal) edge or the chewing (occlusal) surfaces of the teeth, usually on the buccal (vestibular) surfaces and tubercles (Fig. 3, 4).



a ***b***

Figure 4. Enamel hypoplasia:
a — eruption of central incisors with thinned enamel; *b* — eruption of incisors with thinned enamel and enamel aplasia

The hypoplasia of enamel more frequently occurs on the permanent teeth (*K 00.43 Postnatal enamel hypoplasia*), that is related to the diseases of children in the period of forming and mineralization of teeth (approximately from 4.5 months to 2.5–3 years of life). The causes of postnatal enamel hypoplasia are: acute infections, severe form of rickets, toxic dyspepsia, alimentary dystrophy, gastrointestinal pathology, endocrine pathology and other. The older the child is the less labile exchange processes he has. That is why the enamel hypoplasia of premolars and permanent second molars are more rare pathology than hypoplasia of permanent incisors and permanent first molars.

The enamel hypoplasia in infants is rare due to protective function of placenta. Nevertheless, late toxicosis or severe diseases of mother in the second half of pregnancy (german measles, toxoplasmosis and other), causing violation

of integrity of placenta barrier, can also cause such pathology of infant's teeth (usually the incisors). In such cases we are talking about *prenatal enamel hypoplasia* (*K 00.41*).

Difficult birth, disease of child in the first days and weeks of life may cause *neonatal enamel hypoplasia* (*K 00.41*). Such children can have changes of enamel at the necks of primary incisors, middle part of crowns of primary canines and on the masticatory surfaces of primary molars (along the "neonatal line"). The tops of tubercles of the first permanent molars may also be affected.

Hutchinson's teeth and *Fournier's teeth* are kinds of enamel hypoplasia. Such teeth are described in some systemic diseases (for example, Hutchinson's teeth are a sign of congenital syphilis). Their feature is shape like a screw-driver. Hutchinson's teeth also have a half-moon defect on marginal edge (Fig. 5, 6).

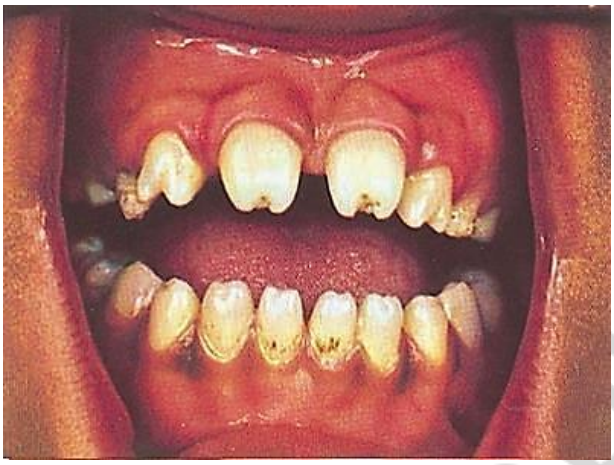


Figure 5. Hutchinson's teeth

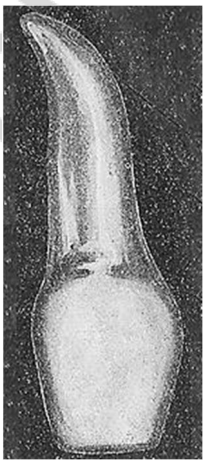


Figure 6. Fournier's tooth

Complaints of patients and their families depend on the clinical form of enamel hypoplasia. Patients with spotted form of enamel hypoplasia complain about the lack of aesthetic. Patients with other forms also complain about a cosmetic defect, but they also can have complaints of pain due to thermal and chemical stimuli. In case of thinning form, cup-like form and enamel aplasia, sharp edges of incisors can damage the labial mucosa. Sometimes break of crowns, teeth erasing can occur. Caries often can develop in the enamel defects.

Differential diagnostics of enamel hypoplasia is carried on with diseases, having a similar clinical manifestations. At diagnosis we usually rely on anamnesis, clinical picture and sometimes on additional methods of diagnosis. That is why careful history taking and patient's dental examination is so important.

Differential diagnostics of the spotted form of enamel hypoplasia is carried on with initial caries, light form of fluorosis, light form of Turner tooth, some types of amelogenesis imperfecta. Solution of "methylene blue" can be used for the differential diagnosis of initial caries and enamel hypoplasia. In case of spotted form of enamel hypoplasia spots are not painted (unlike dental caries).

Differential diagnostics of other form of enamel hypoplasia is carried on with Turner tooth, dentin caries, severe fluorosis, some forms of inherited tooth defects.

The choice of *treatment* depends on:

– the degree of aesthetics disturbance;
– the depth of localization of the defect;
– clinical type of defect and its size;
– posteruptive degree of enamel mineralization;
– the patient's wishes;
– technical capabilities of the doctor.

The method of treatment can be divided into 3 groups (Fig. 7).

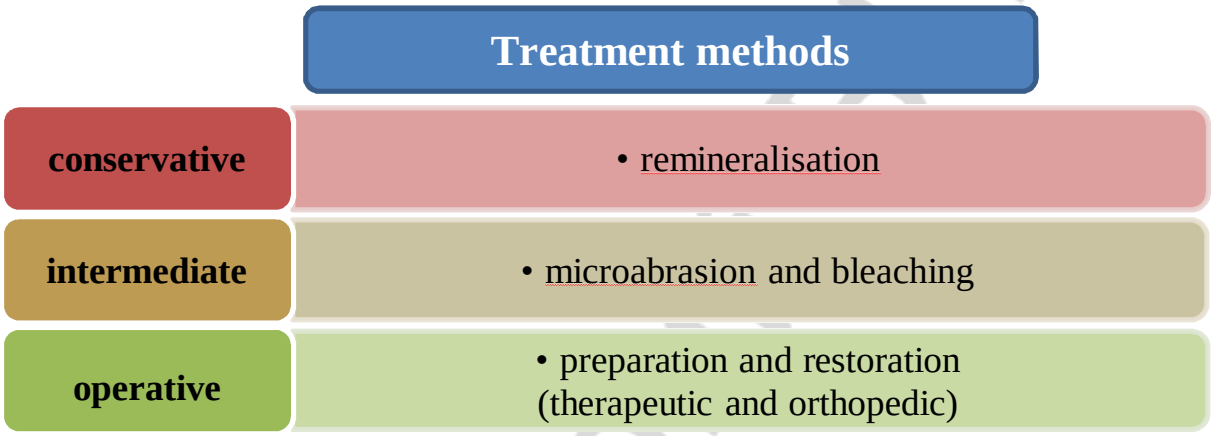


Figure 7. Treatments of enamel hypoplasia

It should be noted that the treatment of enamel hypoplasia in children always starts with motivation and oral hygiene correction to prevent caries development.

Clinical case.

Patient K., girl, 7 years old. Diagnosis — enamel hypoplasia, cup-like form (Fig. 8).

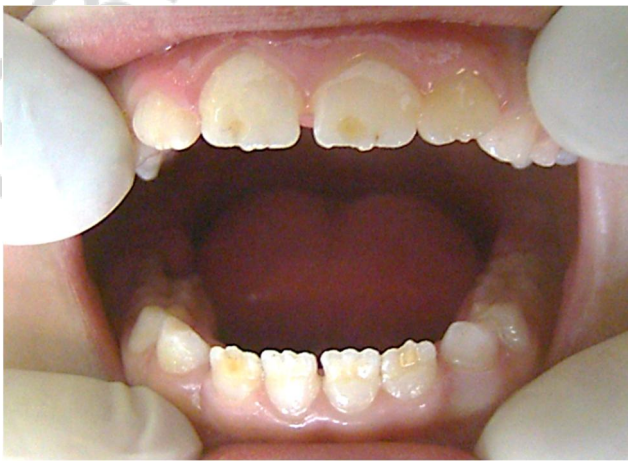


Figure 8. Patient K., 7 years old, restoration in teeth 1.1, initial view

The following treatment plan was made in agreement with the parents of the patient:

– motivation;
– correction of home hygiene;
– diet consultation (talk about a balanced diet including fluoride intake);
– professional hygiene;
– remineralisation;
– temporary GIC restoration (Fig. 9);
– resin restoration after the post-eruptive mineralization.

The *prevention* of enamel hypoplasia is healthy lifestyle, prevention and treatment of somatic diseases of children and their mothers, health of pregnant women.

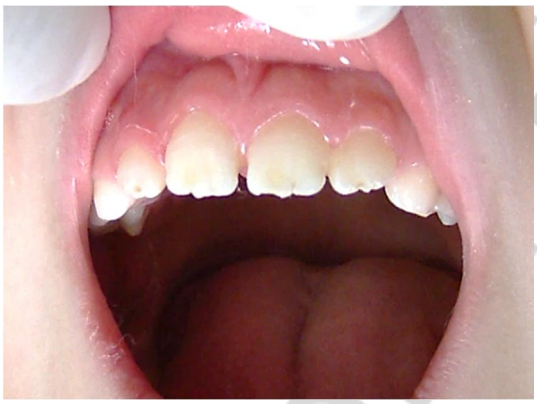


Figure 9. Patient K., 7 years old, after GIC restoration in teeth 1.1, 2.1

К 00.46 TURNER TOOTH

It is a local pathology when one or two teeth (rare) are violated. This teratosis arises up as a result of mechanical trauma of follicle (Fig. 10) or influence of infection (Fig. 11), getting to the permanent tooth follicle.

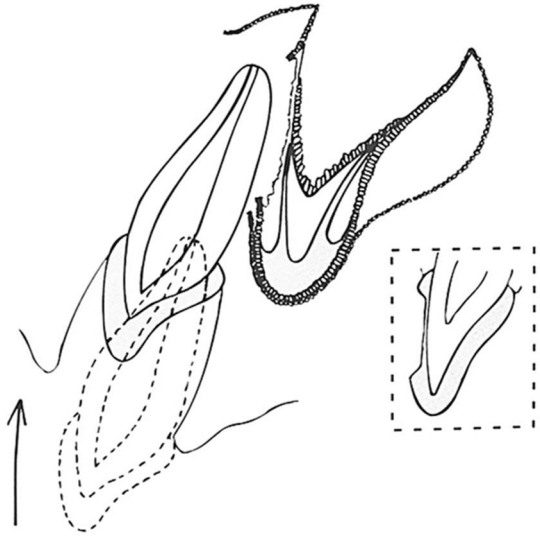


Figure 10. Injury to the permanent tooth bud

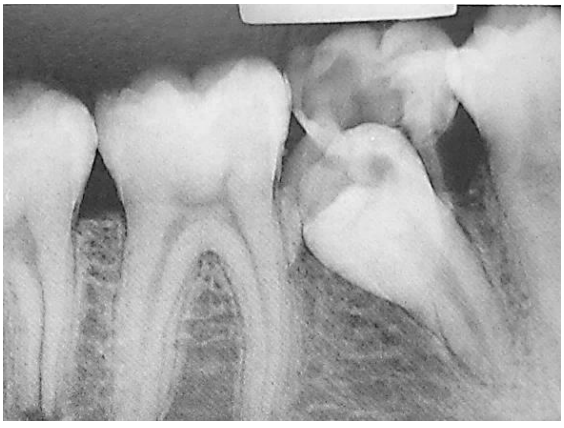


Figure 11 The influence of infection

Turner tooth is a pathology of permanent teeth, it is observed in primary teeth extremely rarely (only in case of osteomyelitis or breakage of a baby's jaw, passing through the primary tooth follicle).The clinical manifestation of Turner tooth largely depends on severity of trauma or inflammatory process and the age of child. The features of turner tooth are the following:

1) spots of various shape, their color is white (rare), yellow or brown, without shining;

2) thinning or aplasia (in severe case) of enamel. The defects have various shapes according to area of damaged ameloblasts. The teeth can be unusual size and form. Caries often appears in the deep defects.

There are 3 clinical forms of Turner tooth according to P. Gaengler (2006):

– discoloration (Fig. 12);

– hypoplasia (Fig. 13);

– combined.

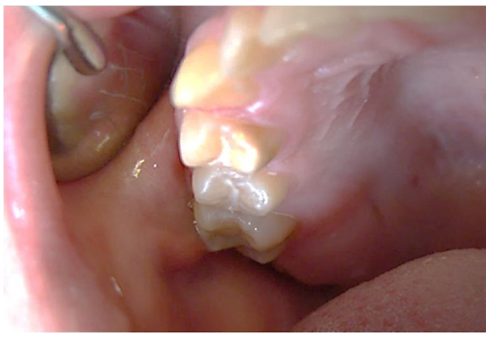


Figure 12. Turner tooth (1.4), discoloration

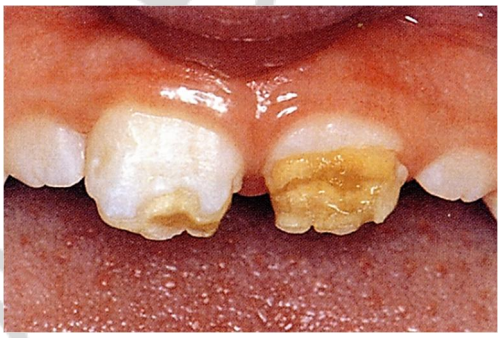


Figure 13. Turner tooth (1.1, 2.1), hypoplasia

Patients *complain* of a lack of aesthetic, sometimes of hypersensitivity from temperature and chemical stimuli.

Differential diagnostics of Turner tooth is carried on with enamel hypoplasia, fluorosis, caries, some forms of inherited tooth defects.

The principles of *treatment* of Turner tooth are the same as for enamel hypoplasia.

The *prevention* of Turner tooth is the prevention of dental trauma in children and prompt and proper treatment of caries of the primary teeth.

К 00.45 REGIONAL ODONTODYSPLASIA

Regional odontodysplasia is an uncommon developmental abnormality of teeth, usually localized in a certain area of the mouth. The enamel, dentin, and pulp of teeth are affected to the extent that the affected teeth do not develop properly. The size of these teeth is decreased, their color is yellow, their surface is rough and their form is changed. These teeth are very brittle, enamel erasing is marked. On radiographs the teeth appear more radiolucent than normal, so they are often described as "ghost teeth" (Fig. 14). Most cases are considered as idiopathic, but some cases are associated with syndromes, growth abnormalities,

irradiation, osteomyelitis, neural disorders, and vascular malformations. Many of affected teeth do not erupt, and those that do have an increased risk of caries and periapical inflammation.

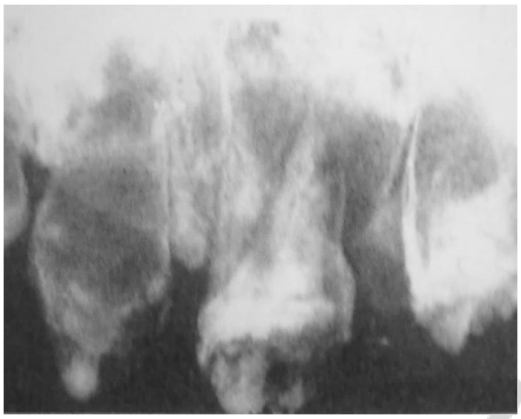


Figure 14. The “ghost teeth”

Differential diagnostics is carried on with enamel hypoplasia, Turner tooth, tetracycline teeth, some inherited tooth defects.

Treatment and prognosis are usually based upon keeping these teeth and preserving the alveolus. For erupted teeth, in mild disease the course of remineralization is recommended. Temporary restorations from GIC, compomers and resin materials can be used. Endodontics also is an option if the tooth is devitalized and restorable.

MOLAR INCISOR HYPOMINERALIZATION

Molar Incisor Hypomineralization (MIH) is an enamel hypomineralization of systemic origin affecting one or more first permanent molars (FPMs) that are frequently associated with affected incisors. The condition is also known as nonfluoride enamel opacities, internal enamel hypoplasia, nonendemic mottling of enamel, opaque spots, idiopathic enamel opacities, enamel opacities, and idiopathic enamel hypomineralization.

A wide variation in the reported prevalence of MIH exists with rates varying from 3.6 % to 37.5 %. Causes of disease are not completely clear, but MIH is significantly more common in children with reported health problems during the first four years of life.

Clinically, the hypomineralized enamel can be soft, porous, or resembling discolored chalk or old Dutch cheese. The enamel defects can vary in color from white to yellow or brown, but they always show a sharp demarcation between the affected and sound enamels. The porous, brittle enamel can easily chip off under masticatory forces. Occasionally, loss of enamel can occur so rapidly after eruption that it seems as if the enamel was not formed initially and giving a picture resembling hypoplasia. The latter, however, has smooth margins to the surrounding enamel, whilst in MIH the borders appear to be irregular.

Molar incisor hypomineralization can sometimes be present with opacities in the upper and lower incisors. The defects of incisors are usually without loss of enamel substance and are generally less serious than those seen in molars due to absence of chewing forces. The second permanent molars and bicuspids are very seldom impaired by these enamel defects. According to Weerheijm et al., the second primary molars, second permanent molars, and the tips of the permanent canines can also show enamel defects occasionally.

Defective molar teeth are more susceptible to plaque accumulation and dental caries and may therefore have a greater need for dental treatment. Because the prismatic morphology in the porous enamel is altered, bonding to enamel becomes difficult leading to frequent loss of fillings and repeated treatment. Children with affected molars generally receive more dental treatments than those without and a significant number of retreated teeth eventually require extraction.

The treatment of teeth with MIH can be painful due to difficulties in anaesthetizing, most likely due to subclinical inflammation of the pulpal cells caused by the porosity of the enamel. Because of the difficulties in achieving adequate anesthesia and frequent treatments, children with hypomineralized first molars may show difficult behavior and dental fear and anxiety. The treatment plan is the same as for enamel hypoplasia.

ENAMEL HYPERPLASIA (K 00.2 ABNORMALITIES OF SIZE AND FORM)

Enamel hyperplasia (“enamel drops”, “pearls”) occurs due to excessive formation of dental tissues. “Enamel drop” can consists of:

– enamel;

– enamel and dentin;

– enamel, dentin and pulp.

Enamel drops are usually 2–4 mm in diameter, rounded form, often located at the neck of tooth (Fig. 15). Treatment (removing excessive tissues) is required only in case of aesthetic disorders or dysfunction. X-ray is required before treatment to predict the possibility of intervention in the pulp.

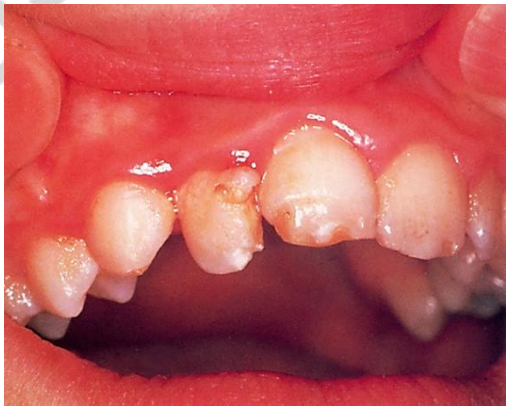
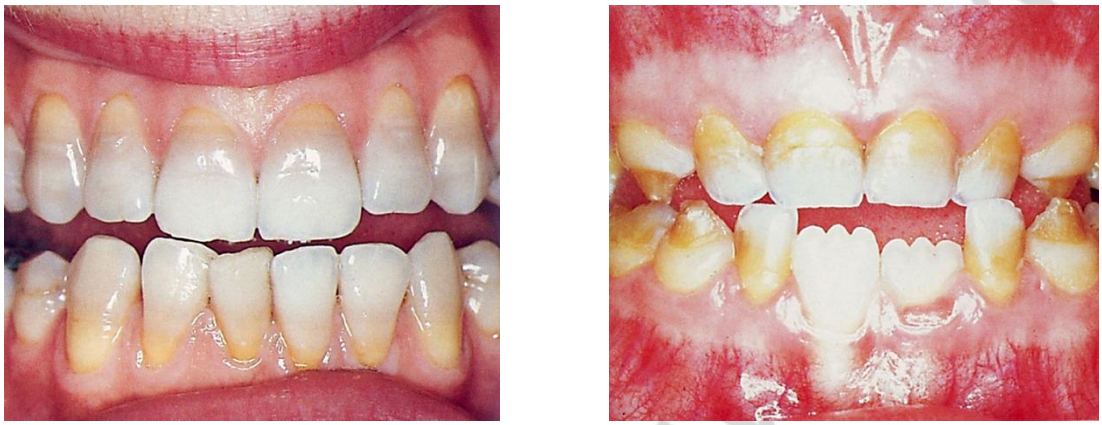


Figure 15. “Enamel drop”

TETRACYCLINE TEETH
(K 00.8 OTHER DISORDERS OF TOOTH DEVELOPMENT)

Tooth discoloration due to receiving tetracycline and its analogues during tooth formation. Not the whole crown is discolored but only its part which was mineralized during receiving tetracycline (Fig. 16, *a*). In severe case we can see dark yellow-grey teeth with a darker horizontal band that goes across the top and bottom rows of teeth. In some cases, tetracycline-stained teeth are not just discolored but also display surface defects (Fig. 16, *b*).



a ***b***

Figure 16. Tetracycline teeth:
a — discoloration; *b* — discoloration and defects

Differential diagnostics is carried on with hemolytic illness of newborn.

The *treatment* of tetracycline teeth in case of mild form includes bleaching in patients over 15 years. Bleaching doesn't work well for severe tetracycline stains and in these cases patients need veneers (resin or porcelain) or artificial crowns after the formation of permanent dentition.

Tetracycline use should be avoided in pregnant or lactating women, and in children with developing teeth crowns (children under 8 years) to *prevent* tooth discoloration (Fig. 17). These categories of patients should receive tetracycline only for vital reasons.

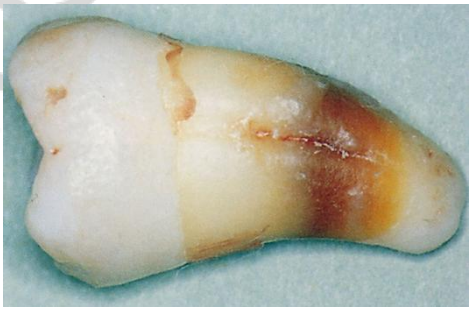


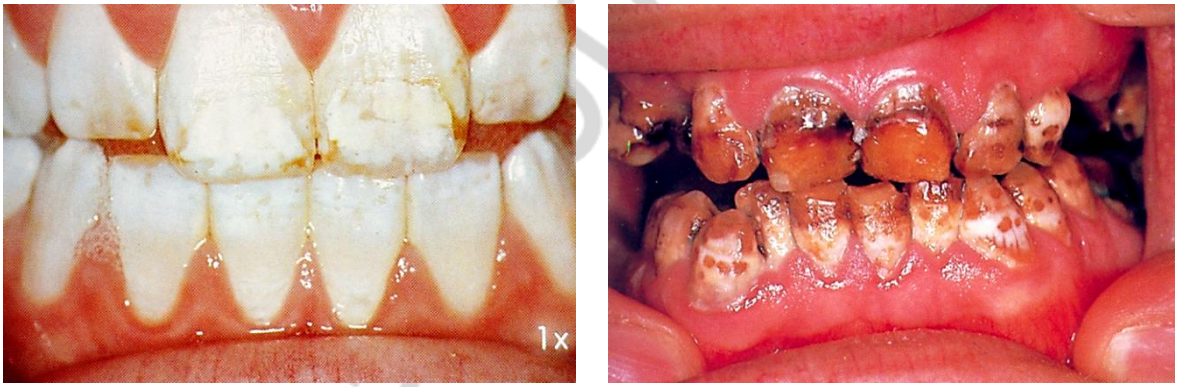
Figure 17. Tetracycline tooth, premolar, discoloration of the root, the crown color is normal. Tetracycline was received in age of 10 years.

DENTAL FLUOROSIS / ENDEMIC MOTTLING ENAMEL (К 00.3 MOTTLED TEETH)

Ingestion of excess fluoride, most commonly in drinking-water, can cause fluorosis which affects the teeth and bones. The dental effects of fluorosis develop much earlier than the skeletal effects in people exposed to large amounts of fluoride.

Fluorosis is endemic in 22 countries around the world. Drinking water containing fluoride is the major cause of fluorosis due to geological crust contamination. The guidelines followed for fluoride content in drinking water in most of the countries are based on the WHO norms. According to the WHO, the desirable upper limit for fluoride in drinking water is 1.5 mg/L.

Clinically dental fluorosis is characterized by staining and pitting of the teeth. In more severe cases all the enamel may be damaged. Due to excessive fluoride intake, enamel loses its lustre. In its mild form, dental fluorosis is characterized by white, opaque areas on the tooth surface (fig. 18, *a*) and in severe form, it is manifestated as yellowish brown to black stains and severe pitting of the teeth (fig. 18, *b*). This discoloration may be in the form of spots or horizontal streaks. Normally, the degree of dental fluorosis depends on the amount of fluoride exposure up to the age of 8–10, as fluoride stains only the developing teeth while they are being formed in the jawbones and are still under the gums.



a ***b***

Figure 18. Dental fluorosis:
a — mild form; *b* — severe form

Dean's Classification System for Dental Fluorosis is commonly used to assess the severity of the pathology. Dean's Index is scored on the condition of the two most severely affected teeth of the patient.

Dean's Classification System for Dental Fluorosis (1942):

Normal (Code 0) — the enamel represents the usual translucent semivitriform type of structure. The surface is smooth, glossy and usually of pale creamy white colour.

Questionable (Code 1) — the enamel discloses slight aberrations from the translucency of normal enamel, ranging from a few white flecks to occasional white spots. This classification is utilised in those instances where a definite diagnosis is not warranted and a classification of “normal” not justified.

Very Mild (Code 2) — small, opaque, paper white areas scattered irregularly over the tooth but not involving as much as approximately 25 per cent of the tooth surface. Frequently included in this classification are teeth showing no more than about 1–2 mm of white opacity at the tip of the summit of the cusps, of the bicuspids or second molars.

Mild (Code 3) — the white opaque areas in the enamel of the teeth are more extensive but do involve as much as 50 percent of the tooth.

Moderate (Code 4) — all enamel surfaces of the teeth are affected and surfaces subject to attrition show wear. Brown stain is frequently a disfiguring feature.

Severe (Code 5) — all enamel surfaces are affected and hypoplasia is so marked that the general form of the tooth may be affected. The major diagnostic sign of this classification is discrete or confluent pitting. Brown stains are widespread and teeth often present a corroded-like appearance.

Differential diagnostics of dental fluorosis is conducted with spotted form of enamel hypoplasia, initial caries, light form of Turner tooth, some types of amelogenesis imperfecta.

Dental fluorosis may or may not be of cosmetic concern. In some cases there may be varying degrees of negative psychosocial effects. The *treatment* options are the following:

– tooth bleaching;
– micro-abrasion;
– resin restorations;
– veneers;
– crowns.

Generally more conservative options such as bleaching are sufficient for mild cases.

Excessive fluoride ingestion by human beings can be *prevented* by using the following approaches:

– using alternate water sources: Alternate water sources include surface water, rainwater and low-fluoride groundwater;

– improving the nutritional status of population at risk: Adequate calcium intake is directly associated with a reduced risk of dental fluorosis. Vitamin C ingestion also safeguards against the risk of fluorosis;

– defluoridation: Removing excess fluoride from drinking water using different techniques such as Nalgonda method. This defluoridation method is based on the combined use of alum and lime in a two-step process.

INHERITED TOOTH DEFECTS. ETHIOPATHOGENESIS, DIAGNOSTICS, CLINICAL PICTURE AND TREATMENT

The cause of all these lesions are different genetic faults.

We can divide inherited tooth defects into:

1) isolated tooth defects (amelogenesis imperfecta, taurodontism, dentinogenesis imperfecta, odontogenesis imperfecta, dentin dysplasia, shell-like teeth);

2) defects combined with somatic pathology (osteogenesis imperfecta, marble bone disease, hypophosphatasia);

3) additional rare defects combined with somatic pathology (Ehlers-Danlos syndrome, Brachium-Skeleton-Genital syndrome, pseudohypoparathyroidism etc.).

AMELOGENESIS IMPERFECTA (K 00.05 HEREDITARY DISTURBANCES IN TOOTH STRUCTURE, NOT ELSEWHERE CLASSIFIED)

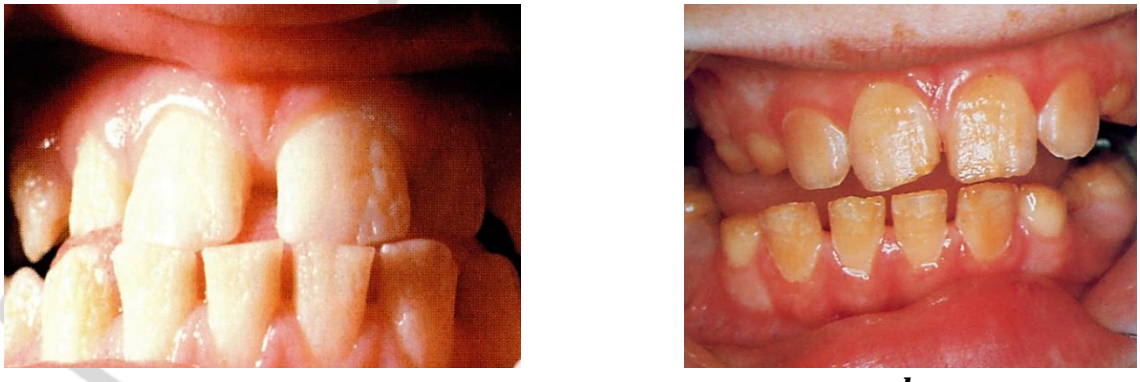
Amelogenesis imperfecta is hereditary defects of development of enamel that affect both the primary and permanent dentition. The defected tooth structure is limited to the enamel. Prevalence of the pathology is 1:14000–1:16000.

There are 4 types of amelogenesis imperfecta according to J. Jr. Witcop (1988):

– Type I (hypoplastic);
– Type II (hypomaturation);
– Type III (hypocalcified);
– Type IV (hypoplastic and hypomaturation)

Type I (Hypoplastic)

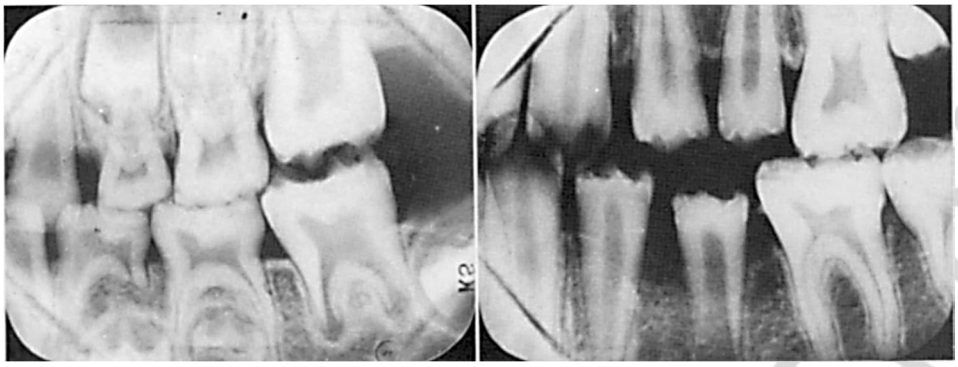
The enamel matrix is imperfectly formed, but calcification occurs in proper way. The enamel is hard but it is defective in amount and has a roughened, pitted surface (Fig. 19) or reduced thickness. Patients / their parents *complain* about a lack of aesthetic.



a ***b***

Figure 19. Amelogenesis imperfecta, type I:
a — pitted surface of enamel; *b* — reduced thickness of enamel

The dentin and pulp are normal. On X-ray examination the pulpal outline and the root morphology are not unlike that of normal tooth (Fig. 20).



a ***b***

Figure 20. Amelogenesis imperfecta, type I, X-ray picture:
a — first transitional period; *b* — second transitional period

Treatment consists of two actions: 1) good oral hygiene to prevent discoloration and caries development (Fig. 21); 2) veneers, artificial crowns in permanent dentition (if patient has complaints due to esthetics disorders).

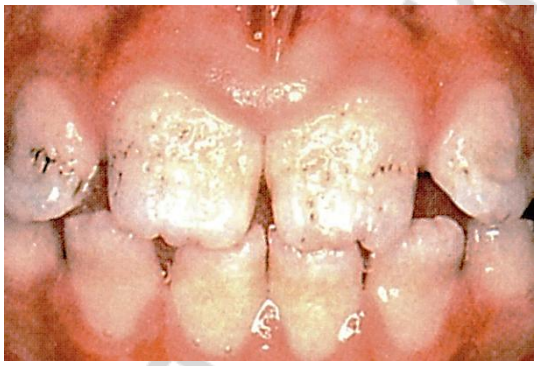


Figure 21. Amelogenesis imperfecta, type I, discoloration and caries in the pits

Type II (Hypomaturation)

The enamel matrix is formed like that of normal tooth, but calcification does not occur in proper way. Normal enamel thickness is observed and the mineral content is less than the norm, but more than in the hypocalcified type. Discoloration is observed on the occlusal part of the crown — from matt white to light amber ("teeth covered with snow"). Post-eruptive enamel chipping can occur (Fig. 22). Pulpal outline and the root morphology are normal.

Complaints of patients and their families are about the lack of aesthetic.

Treatment of the pathology consists of:

– good oral hygiene;

– remineralising therapy;

– halftemporary restoration (if it is necessary);

– veneers, artificial crowns in permanent dentition (if patient has complaints due to esthetics disorders).

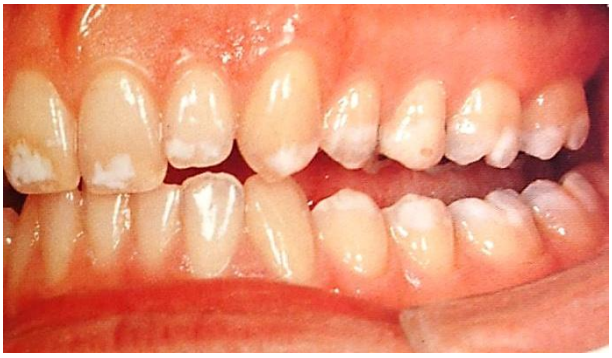


Figure 22. Amelogenesis imperfecta, type II

Type III (Hypocalcified)

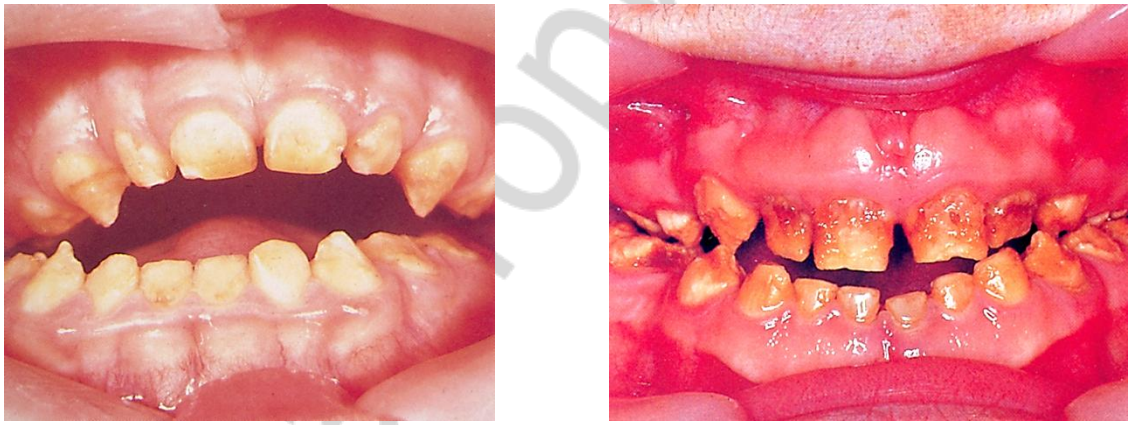
The enamel matrix is formed in proper way, but calcification is deficient. Normal enamel thickness is observed, but the enamel is soft. As a result post-eruptive enamel chipping is observed. Pulpal outline and the root morphology are normal. The teeth erupt normal form (color may be different) but due to enamel chipping:

– abnormal form of crown;

– increased thermal sensitivity (exposed dentin);

– a large amount of dental plaque.

The pathology can be combined with open bite (Fig. 23).



a ***b***

Figure 23. Amelogenesis imperfecta, type III:
a — enamel chipping; *b* — naked dentin and a large amount of dental plaque due to enamel chipping

Complaints of patients and their families are about the lack of aesthetic, hypersensitivity and enamel chipping.

Treatment of the pathology consists of:

– good oral hygine;

– remineralising therapy (in light form and as the initial stage of treatment);

– artificial crowns in primary and permanent dentition (if periodontium is forming – orthodontic crowns).

Type IV (Hypoplastic and Hypomaturation)

A combination of type I and type II. The enamel color is from matt white to yellow and brown and post-eruptive enamel chipping can be observed. The pulp chamber is elongated and extends deeply into the region of the roots (taurodontism).

Complaints of patients and their families are about the lack of aesthetic.

Treatment depends on the severity of clinical manifestations.

TAURODONTISM (K 00.2 ABNORMALITIES OF SIZE AND FORM)

This anomality is characterised by a tendency for the body of the tooth to enlarge at the expense of the roots. The pulp chamber is elongated and extends deeply into the region of the roots (Fig. 24). The prevalence of the pathology is 0.1–3 %.

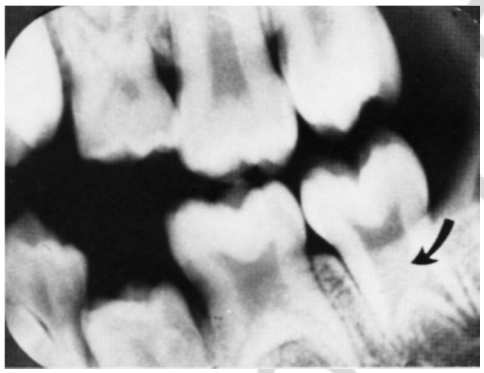


Figure 24. Taurodontism

Patients and their families have *no complaints*, diagnostics is X-ray examination. Taurodontism does not need *treatment*.

Clinical significance of the pathology is difficult endodontic treatment (if it is necessary).

DENTINOGENESIS IMPERFECTA (K 00.05 HEREDITARY DISTURBANCES IN TOOTH STRUCTURE, NOT ELSEWHERE CLASSIFIED)

Dentinogenesis imperfecta is developmental defects of dentin, combined or noncombined with osteogenesis imperfecta. The defected tooth structure is limited to the dentin. The prevalence of the pathology is 1:8000.

Normal dentin formation is confined to a thin layer next to enamel and cementum, followed by a layer of disorderly dentin containing a few tubules and a large amount of water. Enamel-dentin border is a straight line.

There are 3 types of dentinogenesis imperfecta according to Schields (1973):

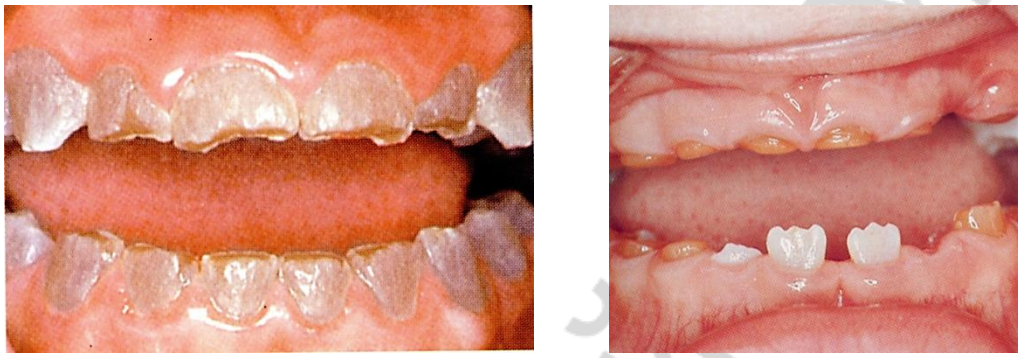
– Type I (in assotiation with osteogenesis imperfecta);

– Type II (isolated pathology);

– Type III (Maryland).

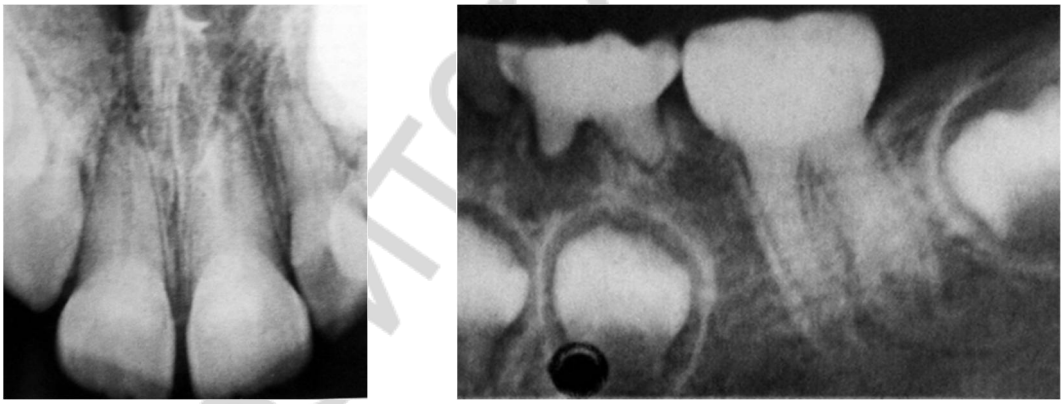
Dentinogenesis imperfecta, type II (isolated pathology, K 00.5 Hereditary disturbances in tooth structure, not elsewhere classified)

The teeth are a characteristic reddish brown to grey opalescence color. Soon after the dentition is complete enamel breaks away from incisal edge (occlusal surface). The exposed dentin abrades rapidly (Fig. 25, *a*). Erasable surface is smooth, the pulp chamber is not opened. The teeth can wear away to the gums (Fig. 25, *b*). The roots are slender and the pulp canals are small and ribbon-like (Fig. 26). There is no satisfactory explanation for absence of pulp necrosis and absence of pulp chamber opening.



a ***b***

Figure 25. Dentinogenesis imperfect:
a — discoloration and erasing of the permanent teeth in adolescent; *b* — eruption of the permanent incisors, the primary teeth are worn to the gums



a ***b***

Figure 26. Dentinogenesis imperfecta, type II, X-ray picture:
a — upper incisors; *b* — molars

Dentinogenesis imperfecta, type I (in assotiation with osteogenesis imperfecta)

Clinical manifestations in the oral cavity are the same as for the type II. There are also triad of extraoral symptoms:

– blue sclera (Fig. 27);
– hearing disorder;
– multiple bone fractures result in reduced growth, bending limbs (Fig. 28).

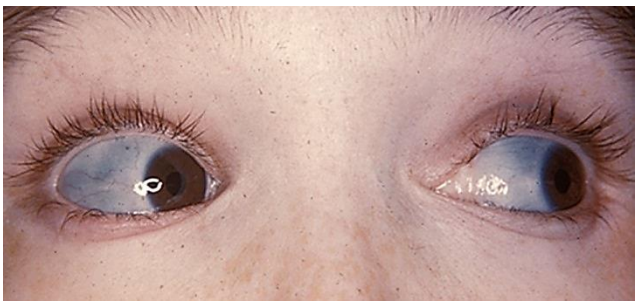


Figure 27. Dentinogenesis imperfecta, type II, blue sclera

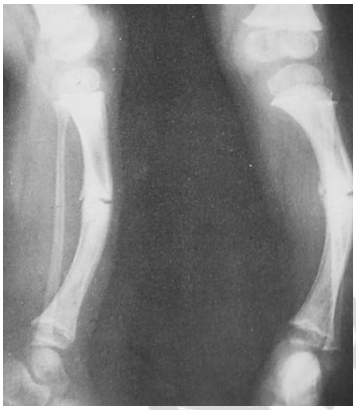


Figure 28. Dentinogenesis imperfecta, bone fractures

Dentinogenesis imperfecta, type III (Maryland) — rare isolated pathology in Maryland, USA as a result of closely related marriages in a small community.

Treatment of the pathology is difficult. Its targets:

– saving of chewing function;

– saving of the teeth bite height;

– erase prevention;

– improving aesthetics.

Possible treatments:

1) during primary dentition — standard and individual crowns on the molars;

2) during permanent dentition: a) covering occlusal surfaces of premolars and molars with resin material; b) after the formation of TMJ — prosthetics and aesthetic restoration; c) protective caps at night after teething.

ODONTOGENESIS IMPERFECTA (К 00.5 HEREDITARY DISTURBANCES IN TOOTH STRUCTURE, NOT ELSEWHERE CLASSIFIED)

Odontogenesis imperfecta is developmental defects of enamel and dentin. Clinic is similar to dentinogenesis imperfecta but dental cavities and root canals are wide, their obliteration does not occur (Fig. 29). As a result pulp opening may occur.

Treatment — artificial crowns for prevention of pulp expose.

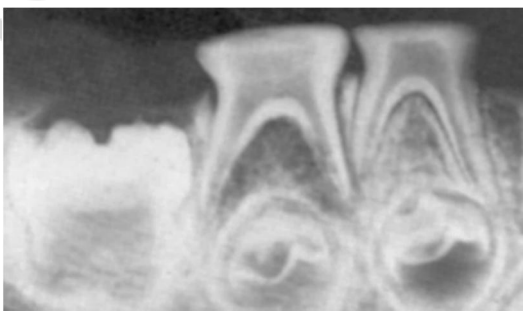


Figure 29. Odontogenesis imperfecta, wide pulp cavity

DENTIN DYSPLASIA (K 00.5 HEREDITARY DISTURBANCES IN TOOTH STRUCTURE, NOT ELSEWHERE CLASSIFIED)

Dentin dysplasia is a rare disturbance of dentin formation. It is characterized by normal formation of enamel, but dentin next to pulp has abnormal structure. Root morphology of temporary and permanent teeth is also abnormal. The prevalence of the pathology is 1:100 000.

There are 2 types of dentin dysplasia according to Schields (1973):

– Type I (radicular dentin dysplasia);

– Type II (coronal dentin dysplasia).

Type I (radicular dentin dysplasia)

Both primary and secondary dentition are affected. The patient has normal color (seldom — opalescent, blue and brown) and morphology of the tooth crowns.

Complaints are of toothache, tooth mobility.

Diagnostic is X-ray examination. The following abnormalities are observed on the X-ray (Fig. 30):

– hypoplasia of the roots (pointed tip);

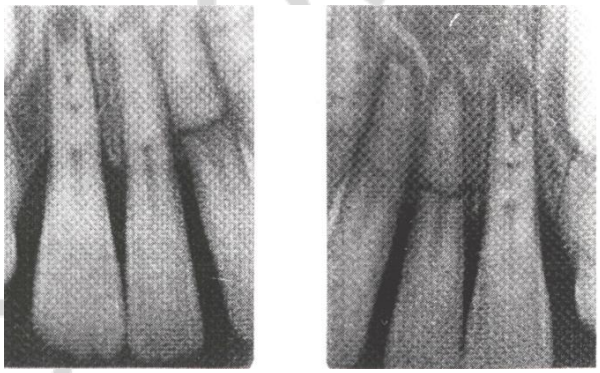
– pulp chamber and tooth root canals are obliterated;

– bleaching line is in the cervical region of roots (fracture);

– periapical changes;

– bone loss (tops interdental partitions are located below the enamel-cement border).

Treatment of the radicular dentin dysplasia is teeth removal and prosthetics.



a ***b***

Figure 30. Radicular dentin dysplasia, X-ray picture:
a — obliterated root canals; *b* — root fractures

Type II (coronal dentin dysplasia)

Both primary and secondary dentition are affected. The primary dentition appears opalescent and on radiographs has obliterated pulp chamber, similar to the appearance of dentinogenesis imperfecta. But unlike in dentinogenesis imperfecta patient has normal color (seldom — light discoloritis) and morphology of the permanent tooth crowns.

Patients and their families have *no complaints*.

The pathology is usually *diagnosed* incidentally during radiographic examinations for other reasons. The following abnormalities are observed on the X-ray (Fig. 31):

– expansion of the coronal cavities (thistle);

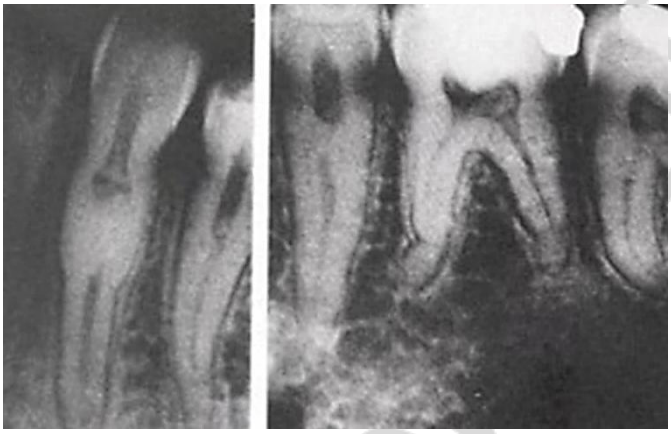
– narrow root canals and deformation of the roots;

– pulp stones;

– the absence of periapical changes.

Coronal dentin dysplasia does not need *treatment*.

Clinical significance of the pathology is difficult endodontic treatment (if it is necessary).



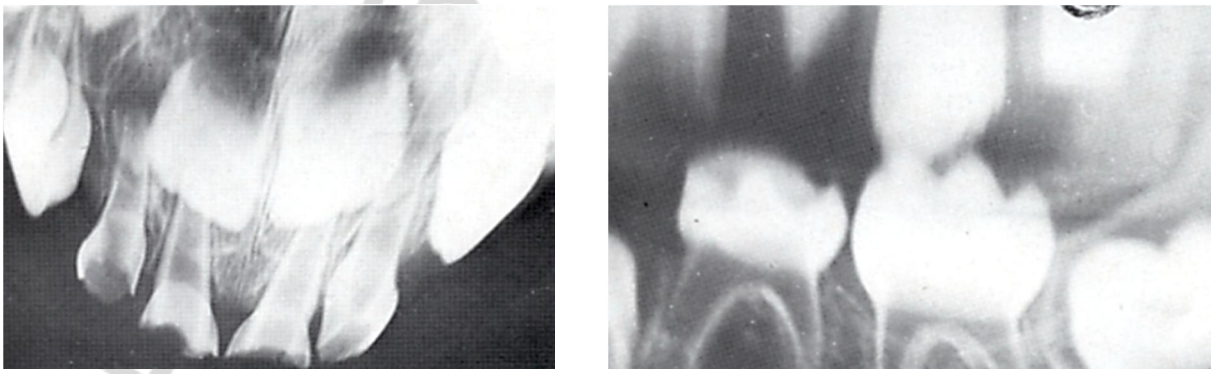
a ***b***

Figure 31. Coronal dentin dysplasia, X-ray picture:
a — lower canine; *b* — lower molars and premolar

SHELL TEETH (К 00.5 HEREDITARY DISTURBANCES IN TOOTH STRUCTURE, NOT ELSEWHERE CLASSIFIED)

The pathology is described Rushton (1954). There is only a thin layer of dentin next to enamel and cementum. Roots are short (Fig. 32).

Treatment of the pathology is not developed.



a ***b***

Figure 32. Shell-like teeth, X-ray picture:
a — primary incisors; *b* — primary molars

MARBLE BONE DISEASE (SYNONYMS: OSTEOPETROSIS, ALBERS-SCHÖNBERG DISEASE)

Marble bone disease is an extremely rare (prevalence is 1:100 000–1:200 000) inherited disorder whereby the bones harden, becoming denser.

The cause of the disease is thought to be malfunctioning osteoclasts. Radiological findings will show a bone-in-bone appearance, marble figure bones.

Mild osteopetrosis may cause no symptoms, and present no problems. Serious forms localized in the maxillofacial region can result in blindness, facial paralysis, and deafness, due to the increased pressure put on the nerves by the extra bone. Periods of exacerbation may be replaced by periods of exacerbation of spontaneous remission due to resorption of newly formed bone. Severe cases may be fatal.

Teeth are of defective quality, enamel hypoplasia, microscopic dentinal defects and arrested root development, retardation of tooth eruption due to the sclerosis of bone all having been reported. Fracture of the jaw during tooth extraction may also occur without undue force.

The *differential diagnoses* include disorders which can cause osteosclerosis, such as hypervitaminosis D and hypoparathyroidism, Paget's disease, intoxication with fluoride, lead or beryllium, et cetera.

Treatment may include gamma-interferon (it delays progression of the disease), bone marrow transplantation, calcitriol (it stimulates osteoclasts to dissolve bone). Dental treatment depends on the clinical manifestations.

HYPOPHOSPHATASIA (SYNONYMS: DEFICIENCY OF ALKALINE PHOSPHATASE, PHOSPHOETHANOLAMINURIA)

Hypophosphatasia is a rare (prevalence is 1:100 000), and sometimes fatal, metabolic bone disease.

Tissue non-specific alkaline phosphatase deficiency in osteoblasts and chondrocytes impairs bone mineralization, leading to rickets or osteomalacia.

Clinical symptoms are heterogeneous, ranging from the rapidly fatal, perinatal variant, with profound skeletal hypomineralization and respiratory compromise, to a milder, progressive osteomalacia later in life. The symptoms largely depend on the age of the patient at initial presentation, ranging from death in utero to relatively mild problems with dentition in adult life. Although several clinical sub-types of the disease have been characterized, based on the age at which skeletal lesions are discovered, the disease is best understood as a single continuous spectrum of severity.

Infantile hypophosphatasia presents in the first 6 months of life, with the onset of poor feeding and inadequate weight gain. Clinical manifestations of rickets often appear at this time. Although cranial sutures appear to be wide, this

reflects hypomineralization of the skull, and there is often “functional” craniosynostosis. If the patient survives infancy, these sutures can permanently fuse. Defects in the chest, such as flail chest resulting from rib fractures, lead to respiratory compromise and pneumonia. Elevated calcium in the blood (hypercalcemia) and urine (hypercalcenuria) are also common, and may explain the renal problems and recurrent vomiting observed in this disease. Mortality is estimated to be 50 % in the first year of life.

Childhood hypophosphatasia has variable clinical manifestations. As a result of defects in the development of the dental cementum, the deciduous teeth (baby teeth) are often lost before the age of 5. Frequently, the incisors are lost first; occasionally all of the teeth are lost prematurely. Dental radiographs can show the enlarged pulp chambers and root canals that are characteristic of rickets.

Patients may experience delayed walking, a characteristic waddling gait, stiffness and pain, and muscle weakness (especially in the thighs) consistent with nonprogressive myopathy. Typically, radiographs show defects in calcification and characteristic bony defects near the ends of major long bones. Growth retardation, frequent fractures, and low bone density (osteopenia) are common. In severely-affected infants and young children, cranial bones can fuse prematurely, despite the appearance of open fontanels on radiographic studies. Premature bony fusion of the cranial sutures may elevate intracranial pressure.

Diagnosis is based on dental finding, laboratory testing, radiography and genetic analysis.

Treatment consists of palliating symptoms, maintaining calcium balance and applying physical, occupational, dental and orthopedic interventions if necessary.

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CONTENT

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