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КАФЕДРА СТОМАТОЛОГИИ ДЕТСКОГО ВОЗРАСТА

Н. В. Шаковец, Ж. М. Бурак, М. И. Кленовская

ЗАБОЛЕВАНИЯ ПЕРИОДОНТА У ДЕТЕЙ

PERIODONTAL PATHOLOGY IN CHILDREN

Учебно-методическое пособие

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Шаковец, Н. В.

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Посвящено проблеме заболеваний периодонта у детей. Освещены вопросы этиологии и патогенеза воспалительных заболеваний периодонта, рассмотрены особенности возрастной нормы, номенклатура болезней периодонта, подробно изложены особенности клинических проявлений различных форм гингивитов, быстропрогрессирующего и симптоматического периодонтитов у детей. Представлены современные подходы к лечению и профилактике заболеваний периодонта у детей.

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ANATOMICAL AND PHYSIOLOGICAL FEATURES OF PERIODONTAL TISSUE IN CHILDREN

PERIODONTIUM STRUCTURE

Periodontium is the tissues that surround and support the teeth, including the gums, cementum, periodontal ligament, and alveolar and supporting bone [5].

It is also called alveolodental membrane, peridental membrane.

Gingiva (or gums) is the part of the oral mucous membrane that covers the alveolar processes and cervical portions of the teeth.

The topography of the gums and the alveolar mucosa is represented on the fig. 1.

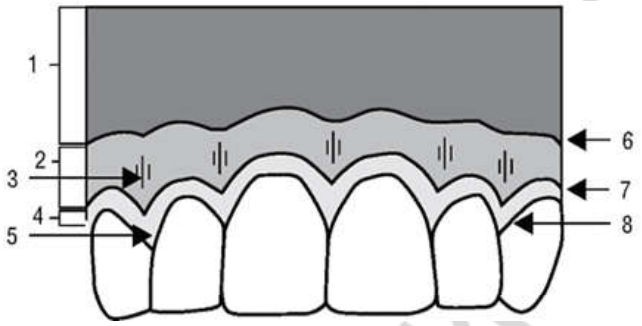


Fig. 1. Cingival topography:
1 — alveolar mucosa; *2* — attached gingiva; *3* — interdental groove; *4* — free gingiva; *5* —papilla; *6* — border between the attached part of the gingiva and the alveolar mucosa; *7* — gingival sulcus; *8* — gingival margin

The gingiva is divided into the papillary portion, which occupies the interdental space, the marginal portion, which forms the collar of free gingiva around the neck of the tooth, and the attached gingiva, which is attached by fibrous tissue to the underlying alveolar bone. Marginal and papillary gingivae together constitute the free gingiva.

Gingival sulcus is also important anatomical structure (it is the space between the free gingiva and the tooth surface). The base of the sulcus is formed by the junctional epithelium — a specialized type of epithelium that attaches to the tooth surface. In case of pathology the junctional epithelium forms the base of periodontal pocket. A periodontal pocket is a sulcus that has deepened because of the disease. The depth of a periodontal pocket is greater than 3 mm.

Attached gingiva extends from the free gingival margin to the mucogingival line minus the sulcus depth in the absence of inflammation. Attached gingiva is necessary to maintain sulcus depth, to resist functional stresses during mastication, and to resist tensional stress by acting as a buffer between the mobile gingival margin and the loosely structured alveolar mucosa.

Healthy gums have the following features:

– coral pink color (although the color may be related to the race identity, age, complexion, the thickness of the tissues and the degree of keratinization).

– gums hug teeth tightly.

– no bleeding.

The periodontal ligament is the connective tissue structure that surrounds the root and connects it with the bone. It is continued with the connective tissue of the gingiva and communicates with the marrow spaces through vascular channels in the bone.

The most important elements of the periodontal ligament are the principal fibers, which are collagenous, arranged in bundles. Terminal portions of the principal fibers that insert into cementum and bone are termed Sharpey's fibers. The principal fibers are arranged in the following groups: transseptal, alveolar crest, horizontal, oblique and apical. Less regularly arranged collagen fibers are found in the interstitial connective tissue between the principal fiber groups which contain blood vessels, lymphatics, and nerves. Other fibers of the periodontal ligament are the elastic fibers, which relatively few, and the so-called oxytalan (acidresistant) fibers, which are distributed mainly around the blood vessels and embedded in cementum in the cervical third of the root.

Cellular elements of the periodontal ligament are fibroblasts, endothelial cells, cementoblasts, osteoblasts, osteoclasts, tissue macrophages, and strands of epithelial cells which are termed the «epithelial rests of Malasser».

Cementum is the calcified mesenchymal tissue that forms the outer covering of the anatomic root.

There are two main forms of root cementum: acellular (primary) and cellular (secondary). Cellular cementum contains cementocytes in individual spaces (lacunae) which communicate with each other through a system of anastomosing canaliculi. Cementoblasts also form the glucoprotein interfibrilary ground substance. Cellular cementum is less calcified than the acellular cementum. It is present only on teeth apices and furcation regions. There are two sources of collagen fibers in cementum: Sharpey's and the fibers belonging to cementum matrix produced by the cementoblasts. Cementum deposition continues throughout life.

Alveolar and supporting bone is the portion of the maxilla or the mandible that forms the dental arch and serves as a bony investment for the teeth.

Functions of the periodontium are the following:

1. *Resistant* — retaining (anatomic) — basic, it is provided by the ligament vehicle of tooth which is in the suspended state. The structure of fibres of periodontium hinders the tissues squeezing and wedging the tooth in the bone of alveolus.

2. *Regulative* (to the division of pressure) — carried out due to a tissue liquid (hydraulic gasket) which is 60 % in periodontium, blood and lymph.

The rich net of blood and lymphatic vessels, operating as a hydraulic system, distributes masticatory pressure evenly on all walls of alveolus and thus improves the function of periodontium as a liaison and amortizing vehicle.

Direction of bunches of collagenous fibres of periodontium answers the direction of forces which operate on teeth at their function exactly. Masticatory effort is physiologically needed for exchange and trophic process in periodontium. At under loading there are changes in the vessels of periodontium that results in disturbance of trophic functions in paradontium in the whole and, results in the development of various pathological processes.

3. *Reflex*. This function is carried out due to a plenty of sensible nervous completions. The force of masticatory pressure is regulated by mechanical receptors from which signals are passed to a masticatory musculature. Liquid environment of periodontium and its fibred structures take part in this process.

4. *Plastic*. Fibroblasts, cementoblasts (build the second cement); osteoblasts (build an alveolar bone) carry out this function.

5. *Trophic*. The net of vessels and nerves provides the normal feeding of tooth cement, compact plate of the alveolus and correct exchange of matters in periodontium.

6. *Sensory*. This function is predefined by the presence of completions of nervous fibers which pierce connective tissue and pass to a peripheral irritation center. Any tooth without pathological changes can perceive touching to the crown and irritation from the particles of food, which get into interdental space.

Normal periodontal condition in primary dentition

The marginal edge of the primary tooth gingiva has a bulky and rounded appearance. The typical stippling in healthy gingiva develops slowly from the age of 2 or 3 years. Marginal gingival tissues around the primary dentition are more highly vascular and contain fewer connective tissue fibers than tissues around the permanent teeth.

The epithelia are thinner with a lesser degree of keratinization giving an appearance of increased redness that may be interpreted as mild inflammation.

Furthermore, the localized hyperaemia that accompanies eruption of the primary dentition persists, leading to swollen and rounded interproximal papillae and the depth of gingival sulcus exceeding 3 mm.

The connective tissue has a similar composition to that around young permanent teeth. However, compared to permanent teeth the primary teeth are associated with a thicker junctional epithelium, which may influence the permeability of the epithelial structures by bacterial toxins. The junctional epithelium of the primary dentition would be less permeable and thus more resistant to inflammation.

On radiographs the alveolar bone surrounding the primary teeth has a distinct but thin, lamina dura and a comparatively wide periodontal membrane. There are few trabeculae and large marrow spaces with rich vascularization.

The root cementum is also thin and mainly cellular.

Alveolar bone has larger marrow spaces, greater vascularity, and fewer trabeculae than adult tissues, features that may enhance the rate of progression of periodontal disease when it affects the primary dentition.

The radiographic distance between the cement-enamel junction and the healthy alveolar bone crest for primary canine and molar teeth ranges from 0 to 2 mm.

Individual surfaces display distances of up to 4 mm when adjacent permanent teeth are erupting or exfoliating, respectively, and eruptive and maturation changes must be considered when radiographs are used to diagnose periodontal disease in children. When such changes are excluded, the distance between cement-enamel junction and alveolar crest more than 2 mm should arouse suspicion of pathological bone loss in the primary dentition.

Normal periodontal condition in mixed and permanent dentition

The exfoliation of primary teeth and the eruption of permanent teeth entail considerable morphological and histological changes.

During eruption of the permanent teeth the junctional epithelium migrates apically from the incisal or occlusal surface towards the cement-enamel junction (CEJ). At this time the gingival sulcus depth often exceeds 6 or 7 mm, which contributes the accumulation of the plaque.

Variation in sulcus depths around posterior teeth in the mixed dentition is common.

Although a periodontal probe is easily inserted deep along the tooth surface, there is no justification for unnecessary explorations interfering with the junctional epithelium.

When the teeth are fully erupted an apical shift of junctional epithelium and free gingival margins are still present. After the tooth is fully erupted, the gingival margin is located on the enamel surface approximately 0.5–2 mm coronal to the cement-enamel junction.

Stability of the gingiva is achieved at about 12 years for mandibular incisors, canines, second premolars, and first molars. The tissues around the remaining teeth continue to recede slowly until about 16 years.

Thus the gingival margins of adjacent teeth are frequently at different levels at different stages of eruption. Sometimes it is mistakenly thought that gingival recession has occurred around those teeth that have been in the mouth longer.

Compared to the primary tooth gingiva a healthy marginal gingiva around permanent teeth is thin and is characterized by pink color.

Table 1

Clinical features of normal periodontium in different age periods

	Period of temporary dentition	Period of mixed occlusion	Period of permanent occlusion
Gum	Gums are bright (increased redness), marginal edge is bulky and rounded, no gingival stippling till the age of 2–3 years.	Gums redness decreases, gingi-val inflammation (eruption gin-givitis) during permanent teeth eruption.	The gum has differentiated mature-suite structure (light pink color, gingival stippling, and triangular papillae).
	Deep gingival sulcus during teething and shallow after teeth eruption.	Deep gingival sulcus during teething (up to 6–7 mm).	The depth of gingival sulcus is up to 3 mm.
	Gingival epithelium is thin, poorly differentiated, with big amount of glycogen, particularly in children under 3 years.	Gingival epithelium thickens, its nipples become more prominent. Glycogen amount decreases.	Gingival epithelium is mature. In the cervical area epithelium is devoid of surface-layer (stratum layer of cells)
	Junctional epithelium is thick and resistant to inflammation.	Junctional epithelium migrates apically during permanent teeth eruption.	Junctional epithelium is thinner and not so re-sistant.
	The basement membrane is thin and delicate.	The basement membrane thick-ens.	The basement membrane is mature.
	Collagen-new fibers are arranged loosely, elastic fibers are absent.	Collagen fibers become denser and navigate.	Collagen fibers are dense and navigate.
	Rich capillary vasculature.	Vascularization decreases.	Mature microvascularization.
	Strong tendency to diffuse reaction	The tendency to diffuse reac-tions is reduced	Normal topical reactivity

End tabl. 1

	Period of temporary dentition	**Period of mixed occlusion**	**Period of permanent occlusion**
Periodontal ligament	Ligament apparatus presents fiber bundles run parallel to the long axis of the tooth and forms an intermediate plexus	Fiber ligament apparatus begins to change its direction, lying at the angle of 45° to the long axis of the tooth	Periodontal ligament is disposed in different directions and tightly fixes the tooth in the alveolus. They mostly come at the angle of 45°, and at the neck of the tooth they are almost horizontal and form a circular bundle
Cementum	Cellular cement is found in the root tip of deciduous teeth and to the period of the change of teeth becomes more powerful	The number of cells increases, and in 10–11 years cellular cement covers about half the length of the roots of temporary and permanent teeth	2/3 of roots is covered with acellular (primary) cement, and the apical third of the roots with secondary (cell) cement
Bone	Lamina dura is distinct but thin. There are few trabeculae, large marrow spaces and rich vascularization.	Alveolar bone becomes more similar with mature bone	Alveolar bone is mature.

The width of attached gingiva is less variable in the primary than in the permanent dentition.

Periodontal ligament space is wider in children, partly as a consequence of thinner cementum and alveolar cortical plates. The ligament is less fibrous and more vascular.

Alveolar bone becomes more similar with mature bone. The radiographic distance between the cement-enamel junction and the healthy alveolar bone crest should be not more than 2 mm (except the period of teething).

Basic clinical features of normal periodontium in different age periods are summed up in tabl. 1.

ETIOLOGY AND PATHOGENESIS OF PERIODONTAL PATHOLOGY

Periodontal disease is an inflammatory process involving progressive, episodic loss of the periodontal attachment apparatus, resulting ultimately in tooth loss in susceptible patients. Periodontal diseases are generally divided into two groups:

1. Gingivitis is an inflammation involving only the gingival tissues next to the tooth.

2. Periodontitis which damages the bone and connective tissue supporting the teeth is a more serious form of gum disease. Periodontitis occurs when the gum tissues separate from the tooth and sulcus, forming periodontal pockets.

Symptoms of the disease. Common symptoms and signs of gum disease include:

– Receding gums;
– Bleeding gums;
– Red, swollen and tender gums;
– Discoloration of gums;
– Formation of spaces between teeth and gums;
– Loose teeth;
– Changes in the way teeth fit together on biting, or the way dentures fit together;
– Continuous bad breath or bad taste in the mouth (halitosis).

Gingivitis is characterized by tender, red, swollen gums that bleed easily and may cause bad breath (halitosis).

Periodontitis is characterized by:

1. Gum inflammation, with redness and bleeding;

2. Deep pockets (greater than 3 mm in depth) are formed between the gum and the tooth;

3. Loose teeth, caused by the loss of connective tissue structures and bone;

Etiology and pathogenesis of periodontal diseases. The main risk factor for the pathology is microorganisms realizing its pathogenic potential in terms of the immune response and environmental conditions. We can say that gum disease develops as a result of plaque buildup because of poor oral hygiene — not brushing and flossing teeth regularly and visiting the dentist.

Factors influencing plaque formation are the following:

– Dental calculus. The surface of the calcified deposits is rough and enhances bacterial colonization, and calculus is therefore deleterious to periodontal health.

– Enamel hypoplasia and hypomineralization. Disturbances in enamel mineralization may lead to a rough surface, which accumulates plaque. The early stages of eruption of hypoplastic teeth may be accompanied by pronounced gingivitis, which disappears later if the cervical part of the tooth has unaffected enamel.

– Dental caries. Obvious carious lesions increase plaque accumulation and gradually impair oral hygiene. Cervical carious lesions are accompanied by a local, chronic gingivitis without exceptions.

– Poor restorations. Restorations with defective margins, rough surfaces or faulty contacts will all cause chronic gingivitis due to increased plaque accumulation.

– Bite pathology. Malocclusion does not play a dominant role in the etiology of periodontal disease, but crowding of teeth may render oral hygiene measures difficult.

– Orthodontic appliances. Fixed orthodontic appliances may impair oral hygiene procedures; bands and brackets accumulate plaque, and removable plates can cause denture stomatitis. Any possible harm to the supporting tissues caused by the appliances must be adequately treated and controlled.

Other risk factors include host factors (problems of immunity; hormonal changes (puberty and pregnancy); somatic diseases (diabetes, hypovitaminosis C, A, E, CNS diseases, hematopoiesis disorders, and gastrointestinal tract diseases), bad habits, etc.) and environmental factors.

Microorganisms can produce disease directly, by invasion in the tissues, or indirectly by bacterial enzymes and toxins. The inflammatory response in periodontal disease includes the activation of leucocytes, neutrophils, T-lymphocytes and plasma cells, the release of antibodies, lipopolysaccharides and chemical inflammatory mediators. The level of periodontal destruction depends on the balance between destructive and protective inflammatory mediators. While periodontal bacteria are required for infective periodontal disease, individual response determines disease progression.

Initial lesion of gums is the result of an inflammatory response to bacterial plaque. It occurs within 2–4 days. The first changes occur around the small gingival blood vessels apical to the junctional epithelium. There is migration and

infiltration of white blood cells into the junctional epithelium and gingival sulcus. There is increased exudation of tissue fluid from gingival crevice. There may be no clinical signs of tissue change at this stage.

If plaque deposition persists, the bacterial plaque becomes older and thicker. The initial inflammatory changes continue with an increased flow of gingival fluid and migration of polymorphonuclear leucocytes (PMNs). There is small increase in the number of inflammatory cells, 75 percent of which are lymphocytes, a few plasma cells and macrophages. There is a breakdown of collagen fiber so that the seal of the marginal cuff of gingiva is weakened.

Early signs of gingivitis become apparent with slight gingival enlargement. Early gingivitis is reversible when plaque is controlled.

Progression from the early lesion leads to the establishment of clinically obvious gingivitis within 7–14 days. Clinical signs of inflammation appear and the interdental papillae may become swollen and bleed on probing. The number of lymphocytes increases and predominant inflammatory cells are plasma cells. Plasma cells are related to areas of chronic inflammation. Clinical signs of inflammation appear and the interdental papillae may become swollen and bleed on probing. Marginal gingiva becomes spongy. With the increased destruction of collagen and inflammatory swelling the gingival margin can be separated easily from the surface giving rise to «gingival» or «false pocket». There is degeneration of junctional epithelium cells and some proliferation of junctional and sulcular epithelium continues. As fibrous tissue is destroyed within the site of active inflammation there is some proliferation of fibrous tissue and formation of new blood vessels at more distant sites. Thus destruction and repair continue side by side.

Continuous plaque irritation and inflammation damages the integrity of the junctional epithelium. There is degeneration and separation of epithelial cells and there is a breakdown of their attachment to the tooth surface. Connective tissue fibers are destroyed. The junctional epithelium proliferates into the connective tissue and down the root surface as the dentogingival fibers and the alveolar crest fibers are destroyed. The epithelium migrates along the root surface. Apical migration of the junctional epithelium continues and as this epithelium separates from the root surface a periodontal or true pocket is formed. The connective tissue is edematous; vessels are dilated and thrombosed; vessel walls break down with hemorrhage into the surrounding tissues. There is a massive inflammatory infiltrate of plasma cells, lymphocytes and macrophages.

The progression of lesion is not prolonged, periods of exacerbation and remission take place and fibrosis is a constant feature. With the destruction of periodontal ligament and alveolar crest resorption, the pocket deepens. Prolongation of the disease may lead to varying degree suppuration and abscess formation. Finally the teeth may become loose, migrate and lost.

CLINICAL AND EPIDEMIOLOGICAL ASSESSMENT OF PERIODONTAL STATE

The diagnosis of gum disease is based on a thorough examination of the patient's mouth by a dentist. The dentist looks for signs of pathology (described above) and assesses the state of periodontium using special indices. In case of periodontitis a full mouth x-ray is taken to determine the extent of the disease.

The calibrated periodontal probe is a periodontal instrument that is marked in millimeter increments and used to evaluate the health of the periodontal tissues. The periodontal probe is the most important clinical tool for obtaining information about the health status of the periodontium.

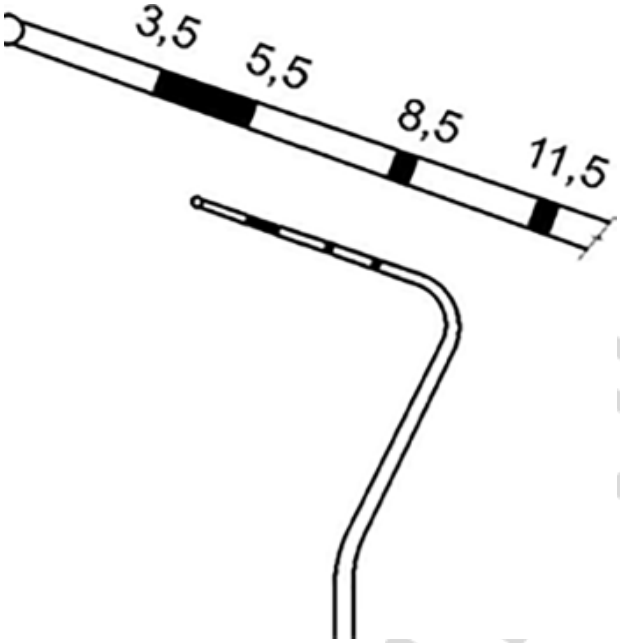


Fig. 2. The calibrated periodontal probe

1. *Design of Calibrated Probes.* Calibrated probes have blunt, rod-shaped working-ends that may be circular or rectangular in cross section. Periodontal probes come in a variety of styles and are made by many different manufacturers. There is a periodontal probe for epidemiological studies conducted by the WHO on the picture. This probe has spherical tip diameter of 0.5 mm and black band (3 mm in length) and 2 rings. As a result the millimeter markings on this particular probe are at 3,5–5,5–8,5–11,5 mm (fig. 2). The probe is used to determine indices recommended by the WHO.

2. Functions of Periodontal Probes. The calibrated periodontal probe is used to measure sulcus and pocket depths, to measure clinical attachment levels, to determine the width of attached gingiva, to assess the presence of bleeding and/or purulent exudate (pus), and to measure the size of oral lesions. In general, the deeper the probe slips into the pockets between a tooth and gums, the more severe the gum disease is in that area.

Indices. There are a number of indices for the assessment of periodontium. The indices may be divided into gingival (PMA, GI) and periodontal (CPI by P. A. Leous, CPITN, Index of gingival attachment loss) (tabl. 2).

Table 2

Simplified scheme of several indices for periodontium assessment

Index	Teeth examined	Surfaces examined	Tools	Scoring criteria	Scoring	Interpretation
PMA	All teeth except third molars	Papillary portion, Marginal portion and Attached gingiva for each tooth	Blunt probe for pressing on gingiva	0 — no inflammation; 1 — inflammation of papillary portion; 2 — inflammation of marginal portion; 3 — inflammation of attached gingiva	Add scores for each tooth and divide by the number of teeth, then multiply result by 100 percent	≤ 33 % — mild gingivitis; 34–66 % — moderate gingivitis; ≥ 67 % — severe gingivitis
GI	16, 21, 24, 36, 41, 44	Buccal, lingual, mesial and distal for each examined tooth	Blunt probe for pressing on gingiva	0 — healthy; 1 — slight discoloration and mild swelling are observed, but bleeding is not observed; 2 — redness, swelling and bleeding are observed; 3 — severe redness, swelling, ulceration and spontaneous bleeding are observed	For each examined tooth add scores for surfaces and divide by 4. Add scores for each tooth and divide by the number of teeth	0 — excellent (no gingivitis); 0.1–1.0 — good (mild gingivitis); 1.1–2.0 — fair (moderate gingivitis); 2.1–3.0 — poor (severe gingivitis)

Continuation tabl. 2

Index	Teeth examined	Surfaces examined	Tools	Scoring criteria	Scoring	Interpretation
CPITN	17/16, 11, 26/27, 47/46, 31, 36/37 as representatives of the appropriate sextants. The index should not be recorded for children under the age of 12. When examining children under the age of 15 years, pockets are not recorded although probing for bleeding and calculus are carried out as routine	Mesiobuccal, buccal, distobuccal, mesiolingual, lingual and distolingual (6 points as a result) for each examined tooth	Calibrated periodontal probe	0 — healthy; 1 — bleeding is observed; 2 — calculus is detected; 4 — pocket ≤ 5 mm is detect-ed; 5 — pocket ≥ 6 mm is detected	Each sextant evaluated on worst score of the index teeth	For each sextant: 1 — there is no need for treatment; 2 — there is need for improving the personal oral hygiene; 3 — there is need for professional hygiene with scaling and root planning; 4 — there is need for complex treatment
Index of gingival attach-ment loss	17/16, 11, 26/27, 47/46, 31, 36/37 as representatives of the appropriate sextants. The index should not be recorded for children under the age of 15	Mesiobuccal, buccal, distobuccal, mesiolingual, lingual and distolingual (6 points as a result) for each examined tooth	Calibrated periodontal probe	0 — loss of attachment 0 to 3 mm; 1 — loss of attachment 4 to 5 mm; 2 — loss of attachment 6 to 8 mm; 3 — loss of attachment 9 to 11 mm; 4 — loss of attachment 12 mm or more	Each sextant evaluated on worst score of the index teeth	Index helps to estimate the level of periodontal attachment destruction

End tabl. 2

Index	Teeth examined	Surfaces examined	Tools	Scoring criteria	Scoring	Interpretation
CPI by P. A. Leous	17/16, 11, 26/27, 47/46, 31, 36/37. The index should not be recorded for children un-der the age of 3 and aged 5–6 years	Mesiobuccal, buccal, distobuccal, mesiolingual, lingual and distolingual (6 points as a result) for each examined tooth	Ordinary dental probe	0 — healthy; 1 — soft deposits; 2 — bleeding is observed; 3 — calculus is detected; 4 — pocket is detected; 5 — loose teeth are detected	Add scores for each tooth and divide by the number of teeth	0.1–1.0 — risk of the disease (pathology); 1.1–2.0 — mild disease (pathology); 2.1–3.5 — moderate disease (pathology); 3.6–5.0 — severe disease (pathology)
Index of gingival	all teeth in the mouth	All surfaces	Visual examina-tion	teeth with gingival recession are determined	the number of teeth with gingival recession is divided intothe number of teeth examined	no

GINGIVITIS IN CHILDREN

Gingivitis is characterized by the presence of gingival inflammation without detectable loss of bone or connective tissue attachment.

Gingivitis in children has the following features:

– similar to adults but with some differences;

– pathological processes develop in a growing, developing and rebuild tissues, that morphologically and functionally are immature and cannot adequately respond to the similar causal factors that cause periodontal disease in adults;

– children have disparity in growing and maturation of immature structures. Imbalance in maturation of structures and formation of functions in the child's body contribute to the development of juvenile gingivitis;

– preschool children tend to be less susceptible to gingivitis than adolescents and adults. The reasons for this difference are not fully understood.

– it has been shown that spirochetes and black-pigmented Bactcroidcs frequently found in adults are not regularly present in the microbial plaque of children with normal gingiva. In addition, the microbial plaque of children with gingivitis has lower proportions of Fusobacterium, Eubacteriunu and Lictobacillus species;

– the increased cell proliferation and turnover of collagen compared with adults may also be significant. The cellular infiltrate of established gingival lesions in children is dominated by T-Lymphocytes and the adult lesion by B-lymphocytes, indicating age-related differences in immunologic response.

– the junctional epithelium in the primary tooth gingiva is thicker than in permanent teeth, therefore it is less permeable to bacterial toxins, and more resistant to inflammation.

The diagnosis of gingivitis is based on the clinical symptoms visible to the eye, such as redness, swelling, and bleeding tendency.

Gingivitis is a reversible disease. Therapy is aimed primarily at reduction of etiologic factors to reduce or eliminate inflammation, thereby allowing gingival tissues to heal. Complete dental care, improved oral hygiene, and supplementation with vitamin C and other water-soluble vitamins will greatly improve the gingival condition. As with all disorders affecting periodontal tissues, maintaining excellent oral hygiene is the primary key to successful therapy. Gingivitis is able to completely disappear under the influence of minimum intervention or independently, and, inspite of the elimination of the reasons to become an independent progressive disease, which in the adult is called «disease of unknown etiology».

Classification of gingivitis in children

Classification of gingivitis in children may be the following:

Acute gingivitis:

– eruption gingivitis;
– herpetic gingivostomatitis;
– necrotizing ulcerative gingivitis.

Chronic gingivitis:

– plaque induced;
– puberty gingivitis.

Gingival enlargement:

– drug-induced (generalized).

Traumatic gingivitis

Mucogingival problems

According to the International Classification of Dental Diseases (1997 г.) classification of gingivitis is the following:

K05 Gingivitis and periodontal diseases

Includes: disease of edentulous alveolar ridge

K05.0 Acute gingivitis

Excludes: acute pericoronitis (K05.22);

acute necrotizing ulcerative gingivitis [fusospirochaetal gingivitis] [Vincent's gingivitis] (A69.10); herpesviral gingivostomatitis (B00.2X)

K05.00 Acute streptococcal gingivostomatitis

K05.08 Other specified acute gingivitis

K05.09 Acute gingivitis, unspecified

K05.1 Chronic gingivitis

K05.10 Simple marginal

K05.11 Hyperplastic

K05.12 Ulcerative

Excludes: necrotizing ulcerative gingivitis (A69.10)

KO5.13 Desquamative

K05.18 Other specified chronic gingivitis

K05.19 Chronic gingivitis, unspecified

K06 Other disorders of gingiva and edentulous alveolar ridge

Excludes: atrophy of edentulous alveolar ridge (K08.2); gingivitis (K05.0, KO5.l)

K06 Other disorders of gingiva and edentulous alveolar ridge

Excludes: atrophy of edentulous alveolar ridge (K08.2); gingivitis (K05.0, KO5.l)

K06.0 Gingival recession

Includes: postinfective

postoperative

K06.00 Localized

K06.01 Generalized

K06.09 Gingival recessions, unspecified

K06.1 Gingival enlargement

Includes: tuberosity

K06.10 Gingival fibromatosis

K06.18 Other specified gingival enlargements

K06.19 Gingival enlargements, unspecified

K06.2 Gingival and edentulous alveolar ridge lesions associated with trauma

K06.20 Due to traumatic occlusion

K06.21 Due to tooth brushing

K06.22 Frictional [functional] keratosis

K06.23 Irritative hyperplasia [denture hyperplasia]

K06.28 Other specified gingival and edentulous alveolar ridge lesions associated with trauma

K06.29 Unspecified gingival and edentulous alveolar ridge lesions associated with trauma

Acute gingivitis in children

Acute gingival inflammation can be caused by a sharp object trauma, high temperature exposure or powerful force during tooth brushing.

Eruption gingivitis is a temporary type of gingivitis. It is often observed in young children when the primary teeth are erupting (fig. 3). The gingival inflammation is related to accumulated dental plaque associated with erupting tooth and it subsides after the teeth emerge into the oral cavity.

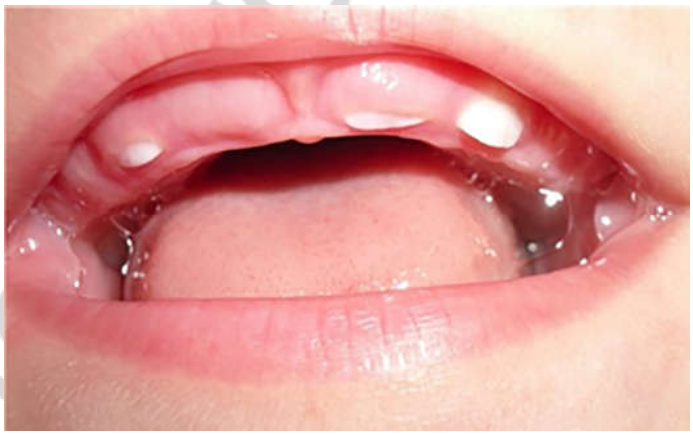


Fig. 3. Eruption gingivitis

The greatest increase in the incidence of eruption gingivitis in children is often seen in 6–7-year old age group when the permanent teeth begin to erupt because the gingival margin receives no protection from the coronal contour of the tooth during the early stage of active eruption, and the continual impingement

of food on the gingiva causes the inflammatory process. Food debris, materia alba and bacterial plaque often collect around and beneath the free tissue, partially cover the crown of the erupting tooth and cause the development of the inflammatory process.

Eruption gingivitis in mixed and permanent dentition is most commonly associated with the eruption of the first and second permanent molars, and the condition can be painful and can develop into pericoronitis or pericoronal abscess.

Treatment. Mild eruption gingivitis requires no treatment other than improved oral hygiene. Pericoronitis accompanied by swelling and lymph node involvement should be treated with antibiotics.

Primary herpetic gingivostomatitis is an acute infectious disease caused by the herpesvirus hominis. Herpetic gingivostomatitis is almost always caused by herpes simplex virus type 1 (HSV-1). The primary infection is most frequently seen in children between 6 months and 5 years of age. The symptoms of the disease develop suddenly and include malaise, irritability, headache and pain associated with an intake of acid food and liquids.

Characteristic oral symptom is the presence of fiery red gingival tissues and yellow or white liquid-filled vesicles (fig. 4). In a few days the vesicles rupture and form painful ulcers, 1–3 mm in diameter, which are covered with a whitish gray membrane with circumscribed area of inflammation. The ulcers may be observed at any area of the mucous membrane including buccal mucosa, tongue, lips, hard and soft palate and tonsillar areas. Large ulcerated lesions may occasionally be observed on the palate or gingival tissues or in the mucobuccal fold region.

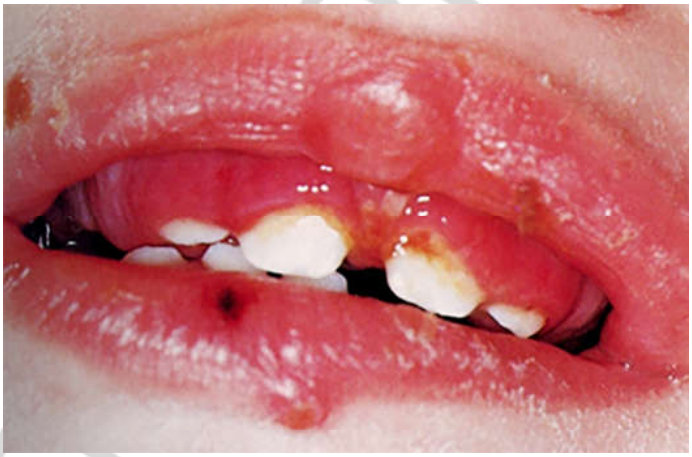


Fig. 4. Primary herpetic gingivostomatitis

Treatment of acute herpetic gingivostomatitis in children, which runs 10–14 days, should include specific antiviral medication as well as relieving acute symptoms to maintain fluid and nutritional intake. The application of mild topical medicine with anesthetic, antiviral, antiseptic and keratoplastic effects are prescribed. Alleviation of generalized symptoms such as fever and malaise is also necessary.

Necrotizing ulcerative gingivitis (NUG; the synonyms are Vincent's disease, fusospirochetal gingivitis, trench mouth, acute ulcerative gingivitis, necrotizing gingivitis, and acute NUG) is one of the commonest acute diseases of the gingiva. In the United States and Europe NUG affects young adults at the age of 16–30 years, incidence figures are 0.7–7 %. In developing countries NUG is prevalent in children as young as 1 or 2 years of age when the infection can be very aggressive leading to extensive destruction of soft and hard tissues.

Epidemic-like occurrences of NUG have been reported in groups such as army recruits and first-year college students.

NUG results from specific microbial flora. Two microorganisms: Borrelia Vincentii and Fusiform bacilli — spirochetal organisms, are frequently associated with NUG. A fusospirochaetal complex has been strongly implicated as the causative organisms in NUG. Other Gram-negative anaerobic organisms including Porphyromonas gingivalis, Veillonella species, and Selenomonas species have been detected, it suggests that NUG could be a broad anaerobic infection.

Predisposing factors of NUG are the following:

– Poor oral hygiene.

– Smoking. Many young adults with NUG are heavy smokers. Smoking may cause local irritation of the gingiva due to the vasoconstrictive action of nicotine, thus reducing tissue resistance and making the host more susceptible to anaerobic infection.

– Food deficiency. In underdeveloped countries children are often undernourished and debilitated which may predispose the infection.

– Stress. It is an important predisposing factors. Elevated plasma level of corticosteroids as a response to an emotional upset is thought to be a possible mechanism.

It is conceivable that all predisposing factors have a common action to initiate or potentiate a specific change in the host such as lowering the cell-mediated response.

NUG is characterized by necrosis and ulceration, which first affect the interdental papillae and then spread to the labial and lingual marginal gingiva (fig. 5, 6). The ulcers are «punched out», covered with a yellowish-grey pseudomembrranous slough, and extremely painful to touch. Acute exacerbation is often superimposed upon pre-existing gingivitis, and the tissues bleed profusely on gentle probing. The standard of oral hygiene is usually very poor. A distinctive halitosis is common in established cases of NUG, although fever and lymphadenopathy are less common than in herpetic gingivostomatitis.

An acute stage of NUG enters a chronic remission in 5–7 days. Recurrence of acute condition is inevitable, however, and if this acute-chronic cycle is allowed to continue then the marginal tissues lose their contour and appear rounded. Eventually,

the inflammation and necrosis involve the alveolar crest and subsequent necrotizing periodontitis leads to rapid bone resorption and gingival recession.

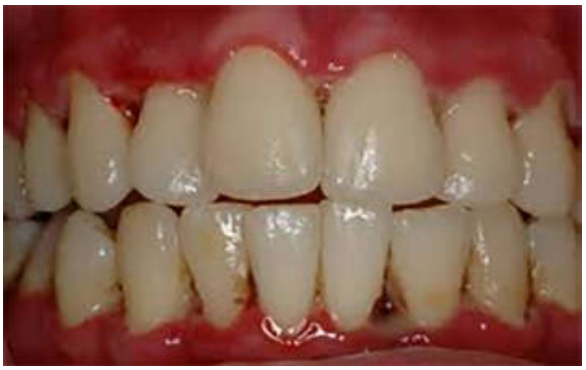


Fig. 5. NUG. Case 1

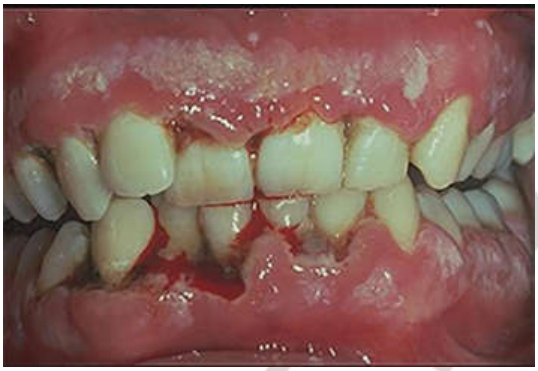


Fig. 6. NUG. Case 2

Treatment. Patient should be informed about the nature of NUG and the likelihood of its recurrence if the treatment is not completed.

Smokers should be advised to reduce the number of cigarettes.

A soft, multitufted brush is recommended when a medium-textured brush is too painful. Mouth rinses may be recommended but only for short-term use (7–10 days). Rinsing with chlorhexidine (0.2 % for about 1 min) reduces plaque formation, while the use of a hydrogen peroxide or sodium hydroxyperborate mouthrinse oxygenates and cleanses the necrotic tissues.

Mechanical debridement should be undertaken at the initial visit. An ultrasonic scaler with its accompanying water spray can be effective with minimal discomfort for the patient. If NUG is localized to one part of the mouth, local anaesthesia of soft tissues can allow subgingival scaling to be undertaken.

In severe cases of NUG, a 3-day course of metronidazole (200 mg 3 times a day) alleviates the symptoms, but the patients must be informed that they are required to come for further treatment.

Occasionally, it is necessary to contour the gingival margin surgically (gingivoplasty) to improve tissue architecture and facilitate subgingival cleaning.

Streptococcal gingivostomatitis is caused by bacterial infection (Streptococcus viridans, group A ß-hemolytic streptococcus). It is characterized by diffuse erythema of the gingiva and other areas of the oral mucosa without gingival necrosis, and fetid odor. Bacterial smears should be used to confirm the diagnosis.

Treatment includes alleviation of acute oral symptoms by reducing microbial load and correction of systemic conditions that contribute to the initiation or progression of gingival changes.

Gonococcal stomatitis is caused by Neisseria gonorrhoeae. It is most common in new born due to transmission through maternal passages. Oral mucosa is covered with a grayish membrane that sloughs off in areas to expose an underlying raw bleeding surface.

Treatment includes alleviation of acute oral symptoms by reducing microbial load and correction of systemic conditions that contribute to the initiation or progression of gingival changes.

Chronic gingivitis in children

Chronic gingivitis must be regarded as a multifactorial disease and a number of intrinsic as well as extrinsic factors influence the severity of its manifestation. As in adults, gingivitis and periodontitis in children and adolescents are primarily caused by accumulation of bacterial plaque on the teeth.

Severe gingivitis is relatively uncommon in children and may also signal the presence of a general disorder, especially if the gingival inflammatory reaction is out of proportion to the amount of bacterial plaque present.

Chronic marginal gingivitis is the most common form of periodontal disease in children and begins in early childhood.

The gingivitis is caused by microbial plaque. Streptococcus intermedius, Streptococcus sanguis, Actinomyces odontolythicus, Actinomyces naeslundii and Veilonella parvula are identified in individuals with simple marginal gingivitis.

The gingiva exhibits all signs of chronic inflammation. The marginal gingiva becomes reddish, with a swollen appearance and papillae protruding from the interproximal spaces. The volume is increased and the surface is shiny. Crevicular exudation is clinically obvious, especially when light pressure is applied to the free gingiva. There is also an increased tendency towards gingival bleeding on probing. Color change and swelling are more common in children than bleeding or increase in pocket depth (fig. 7).

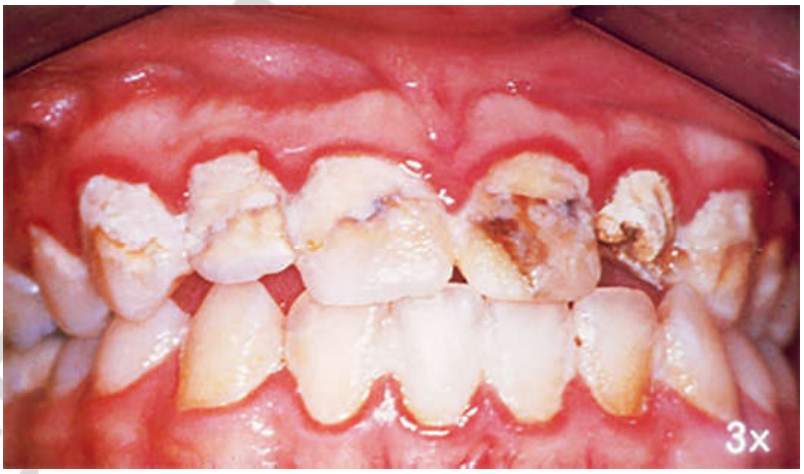


Fig. 7. Chronic simple marginal gingivitis

Treatment stages are the following:

1. Maintenance of individual oral hygiene;

2. Professional oral hygiene;
3. Dental treatment;
4. Treatment of malocclusion;
5. Correction of orthodontic devices;
6. Surgery (frenotomy and frenectomy, vestibular depth increase).

Inflammatory reactions in the marginal gingiva should primarily be regarded as a natural defense against microorganisms. Since the causative factor is plaque accumulation, an efficient oral hygiene regimen will eliminate clinical symptoms rapidly. However, a new period of poor oral hygiene will result in the recurrence.

Desquamative gingivitis is not a specific disease entity (the etiology is unclear), but a gingival response associated with a variety of conditions, such as:

a. Dermatoses (Oral lichen planus, Mucous membrane pemphigoid, Pemphigus vulgaris, Bullous pemphigoid, Erythema multiforme, Linear IgA disease, Lupus erythematosus, Epidermolysis bullosa aquisita, Dermatitis herpetiformis);

b. Local hypersensitivity reactions to toothpastes, mouthwashes, dental materials, drugs, cosmetics, chewing gum, cinnamon, etc.;

c. Miscellaneous (Chronic ulcerative stomatitis, Orofacial granulomatosis, Plasma cell gingivitis).

Females are more frequently affected mainly at the middle and advanced age.

Patients complain of soreness, especially when eating spicy or acidic food and bleeding and discomfort while tooth-brushing. Warmth, tenseness, tingling, itchiness, burning, and pain can be among complaints.

Desquamative gingivitis is characterized by intense erythema, desquamation and ulceration of free and attached gingiva. Vestibular aspect of anterior gingiva is more commonly affected. The gingiva is fiery red, friable and desquamates easily. Sometimes formation of hemorrhagic bullae occurs due to pressing. Lesions get aggravated by local plaque accumulation (fig. 8).

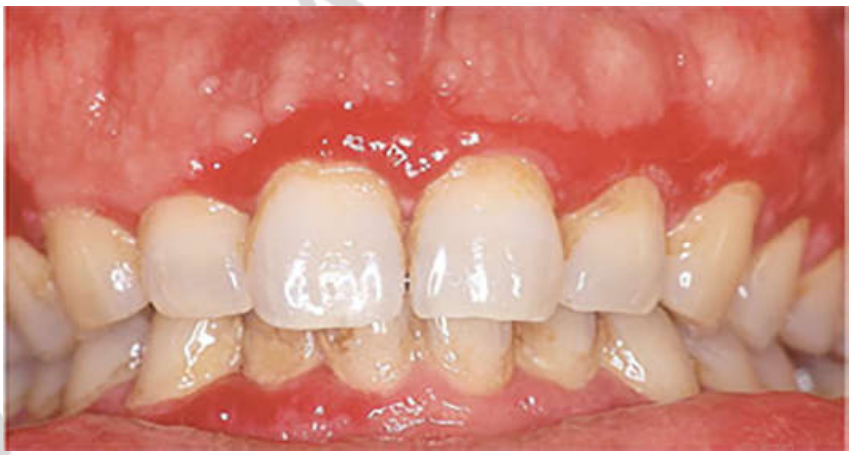


Fig. 8. Desquamative gingivitis

Treatment includes:

1. Maintenance of individual oral hygiene;
2. Stimulants (spicy foods, etc.) Avoidance;
3. Identification and management of the contributing conditions in collaboration with other clinicians (for example systemic corticosteroids in pemphigus);
4. Local treatment:

– mouth washing with 3% diluted to 1/3 peroxide and 2/3 warm water twice a day;

– topical corticosteroid ointments (Triamincinolone 0.1% (Kenalog, Aristocort), Flucocinonide 0.5% (Lidex), Desonide 0.5% (Tridesilon)) in lichen planus and mucous membrane pemphigoid. It should be applied directly onto the affected gingiva and has limited success.

Hyperplastic gingivitis is an increase of the gingiva in size, it is also termed as gingival enlargement or gingival overgrowth.

According to the etiologic factors and pathologic changes hyperplastic gingivitis is divided into:

1. Inflammatory enlargement:
   a) Chronic;
   b) Acute.
2. Drug induced enlargement.
3. Enlargements associated with systemic diseases:
   a) Conditioned enlargement:
      – Pregnancy;
      – Puberty;
      – Vitamin C deficiency;
      – Plasma cell gingivitis;
      – Nonspecific conditioned enlargement.
   b) Systemic diseases causing gingival enlargement^
      – Leukemia;
      – Granulomatous diseases (Wegener's granulomatosis, sarcoidosis).
4. Neoplastic enlargement:
   a) Benign tumors;
   b) Malignant tumors.
5. False enlargement:

According to the location and distribution hyperplastic gingivitis is divided into:

a) Localized: Limited to the gingiva adjacent to a single tooth or group of teeth (the gingival enlargement localized in the canine region);

b) Generalized involving the gingiva throughout the mouth.

Scoring of Hyperplastic gingivitis is the following:

Mild — less than one third of the clinical crown is covered;

Moderate — from one third to two thirds of the clinical crown is covered;

Severe — more than two thirds of the clinical crown are covered.

The most common kinds of hyperplastic gingivitis in children are puberty gingivitis, hereditary gingival fibromatosis, Phenytoin induced gingival overgrowth and ascorbic acid deficiency gingivitis.

Puberty gingivitis is a sample of gingival enlargement associated with the endocrine system. It occasionally develops in children in the pre-pubertal and pubertal period — 11–14-year age group.

The enlargement of gingival tissues is confined to the anterior segment and may be present in only one arch. The lingual gingival tissue generally remains unaffected.

The gingival enlargement is characterized by prominent bulbous interproximal papillae in the presence of local irritants (fig. 9).

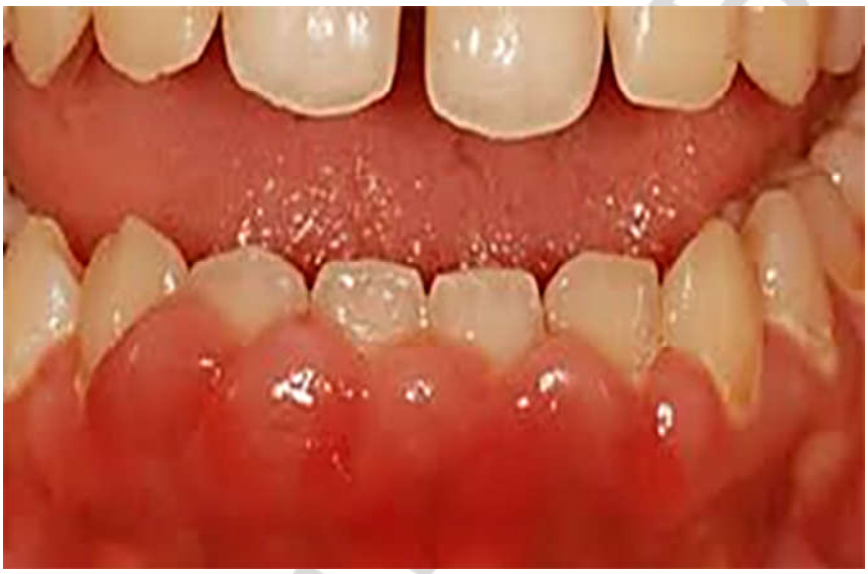


Fig. 9. Puberty gingivitis

Treatment of puberty gingivitis should be directed toward improvement of oral hygiene, removal of all local irritants, adequate nutritional status. Severe cases of hyperplastic gingivitis that do not respond to local or systemic therapy should be treated by gingivoplasty.

Hereditary gingival fibromatosis (HGF) is also known as elephantiasis gingivae or hereditary hyperplasia of the gums. It is of genetic origin (autosomal dominant mode of inheritance usually) and is characterized by a slow, progressive, benign enlargement of the gingivae.

Gingival tissues appear normal at birth but begin to enlarge with the eruption of the primary teeth and continue to enlarge with the eruption of the permanent teeth until the tissues essentially cover clinical crowns of the teeth. Dense fibrous tissue often causes displacement of the teeth and malocclusion. The condition is

not painful until the tissue enlarges to the extent that it partially covers the occlusal surface of the molars and becomes traumatized during mastication (fig. 10).

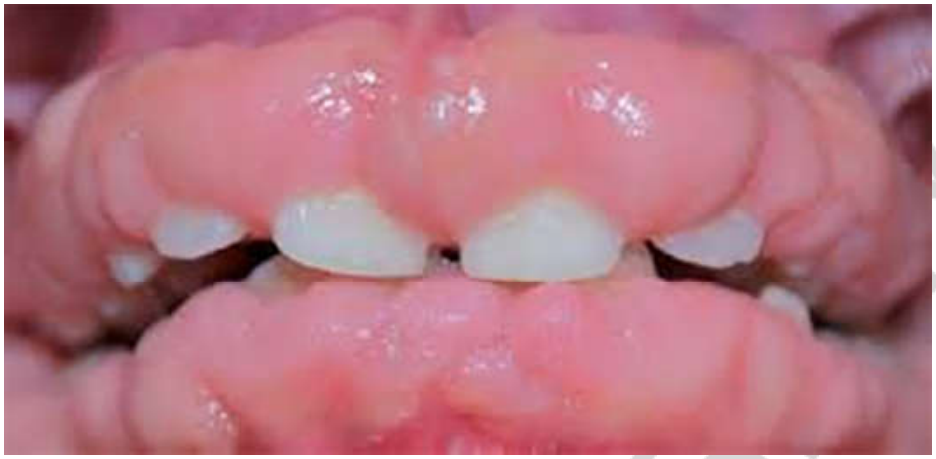


Fig. 10. Hereditary gingival fibromatosis

Treatment is a surgical removal of the hyperplastic tissue. The disease can recur within a few months after the surgical procedure.

Drug induced gingival enlargement can be caused by anticonvulsants, immunosuppressants and calcium channel blockers usage.

Varying degrees of gingival hyperplasia is one of the most common side effects of *phenytoin* (the major anticonvulsant agent used in epilepsy management) therapy. Gingival enlargement occurs in about 50 % of subjects who are taking the drug, and is most severe in teenagers and those who are in medical institutions. *Phenytoin induced gingival overgrowth (PIGO)* is a typical example of the drug induced gingival enlargement. The exact mechanism by which phenytoin induces enlargement is unclear. The gingival enlargement reflects an overproduction of collagen (rather than a decrease in degradation), and this may be brought about by the action of the drug on phenotypically distinct groups of fibroblasts that have the potential to synthesize large amount of protein. Phenytoin-induced enlargement has been associated with a deficiency of folic acid, which may lead to impaired maturation of oral epithelia.

Other drugs that have been reported to induce gingival overgrowth in some patients include Cyclosporin, Nifedipine, valproic acid, and Phenobarbital.

Most investigators agree on the existence of a close relationship between oral hygiene and gingival enlargement rather than doses of phenytoin. The relationship between plaque, local irritants, and hyperplastic gingivitis is also supported by the observation that patients without teeth almost never develop gingival enlargement. First signs of the gingival pathology appear as early as 2–3 weeks after initiation of phenytoin therapy.

Patients complain of esthetics, difficulty in mastication, delayed tooth eruption, difficulties and/or pain while eating, etc.

The initial clinical appearance is painless enlargement of the interproximal gingiva; it becomes more generalized later. These lesions may remain purely fibrotic in nature (if oral hygiene is good) or may be combined with a noticeable inflammatory component (fig. 11). In some cases, the entire occlusal surface of the teeth becomes covered.

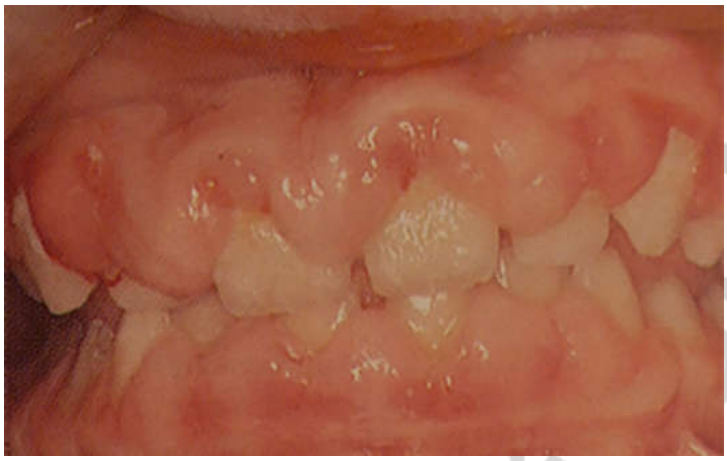


Fig. 11. Drug induced gingival enlargement

The treatment is often symptomatic. Phenytoin levels should be checked in every patient after four prophylaxis visits (4 weeks). If there has been no change, consultation with the patient's physician concerning the possibility of using a different anticonvulsant drug may be helpful.

Treatment according to the severity of PIGO is represented in the table 3.

Table 3

PIGO treatment modalities

Enlargement degree	Treatment modality
Mild	meticulous home oral care
Moderate	meticulous home oral care antiplaque mouthwash 4 consecutive weekly dental office visits for prophylaxis and 5th week — evaluation of the gingiva if no improvement is observed surgical correction is required
Severe	surgical correction and meticulous home oral care

As the causative drugs need to be taken on a long-term basis recurrence is common. When a phenytoin-induced enlargement is refractory to long-term treatment, the patient's physician may be requested to modify or change the anticonvulsant therapy to drugs such as sodium valproate or carbamazepine, which do not cause gingival problems. But cyclosporin, for example, does not have an alternative medication and the patients treated with it inevitably require indefinite oral care.

Ascorbic acid deficiency gingivitis occurs due to lack of vitamin C and differs from the gingivitis related to poor oral hygiene.

The periodontal involvement is usually limited to the marginal tissues and papillae (fig. 12). Severe pain and spontaneous hemorrhage will be evident.

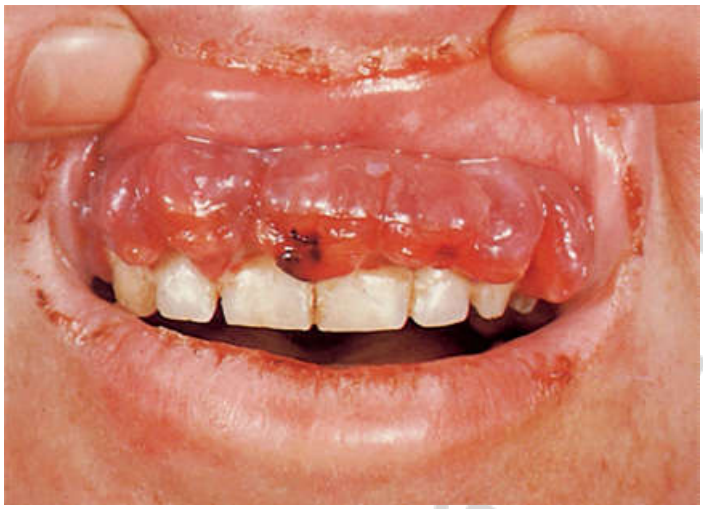


Fig. 12. Ascorbic acid deficiency gingivitis

Treatment includes complete dental care, improved oral hygiene, and supplementation with vitamin C and other water soluble vitamins.

Traumatic gingivitis (Gingivitis artefacta, Factitial gingivitis) has minor and major variants. The minor form results from rubbing or picking the gingiva using the fingernail, or perhaps from abrasive foods such as crisps, and the habit is usually provoked by a locus of irritation such as an area of persistent food packing or an already inflamed papilla. The lesions resolve when the habit is corrected and the source of irritation is removed.

The injuries in gingivitis artefacta major are more severe and widespread and can involve the deeper periodontal tissues. Other areas of the mouth such as the lips and tongue may be involved and extraoral injuries may be found on the scalp, limbs, or face (factitious dermatitis). The lesions are usually viewed with complete indifference by the patient who is unable to remember details of their onset time or possible causes. ***Treatment*** of these patients is not carried out by the dentist, except the dressing and protection of oral wounds. Psychological reasons for inflicting the lesions may be complex and obscure. A psychological or psychiatric consultation, rarely welcomed either by older children or their parents, it is necessary if the patient is to be prevented from ultimately inflicting serious damage upon themselves.

Gingival recession in children

It is a non-inflammatory disease characterized by the displacement of the gum edge in the apical direction (fig. 13).

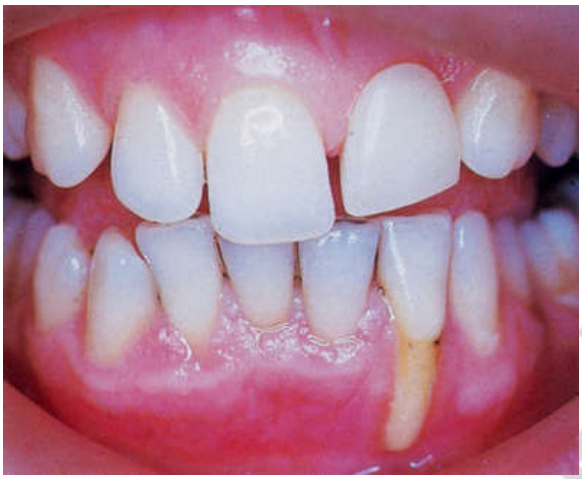


Fig. 13. Gingival recession

Anterior teeth with narrow zones of keratinized gingiva (KG) are frequently encountered in children, as the width of KG varies greatly during the mixed dentition. The width of KG alone should not be the sole indicator of potential sites of gingival recession in children. The position of a tooth in the arch is a better guide as studies have shown that, about 80 % of permanent incisors with recession are displaced labially. Aggravating factors such as gingivitis or mechanical irritation from excessive and incorrect tooth-brushing increase the likelihood of the recession.

Gingival recession is also a common periodontal complication of orthodontic therapy when labial tipping of incisors is undertaken. When roots move labially through the supporting envelope of alveolar bone the potential for recession increases.

AGGRESSIVE PERIODONTITIS IN CHILDREN AND ADOLESCENTS

Aggressive periodontitis (early-onset periodontitis / EOP) is a generic term to describe a heterogeneous group of periodontal disease occurring in younger individuals who are healthy. It is characterized by the rapid loss of periodontal attachment and tooth-supporting bone in healthy patients. EOP is an urgent problem of Periodontology. Aggressive periodontitis prevalence is 0,53–2,5 %.

It is a pathological manifestation of the host response against bacterial challenge caused by a microbial biofilm (dental plaque) at the biofilm–gingival inter-

face. In other words aggressive periodontitis is an inflammatory and infectious process that has an autoimmune nature.

Its features are the following:

– rapid loss of alveolar bone;
– disorders of neutrophil chemotactic function in the peripheral blood;
– decrease of the ratio of T-helper and T-suppressor;
– decreased local protection;
– disturbance of the structure of the connective tissue and microvasculature;
– frequent exacerbations and small periods of remission.

Diagnostic criteria to distinguish chronic and aggressive periodontitis are represented in the tabl. 4.

Table 4

Diagnostic Criteria to Distinguish Chronic and Aggressive Periodontitis (according to the International Workshop for the Classification)

Criterion	Aggressive Periodontitis	Chronic Periodontitis
Rate of prog-ression	Rapid	Slow, but rapid episodes are possible
Familiar aggre-gation	Typical	Just when families share imperfect oral hygiene habits
Presence of etiological factors	Often minimal	Often commensurate with observed periodontal destruction
Age	Often in young patients (i. e., < 35 years old) but can be found in all age groups	Often in older patients (i. e., > 55 years old) but can be found in all age groups
Clinical inflam-mation signs	Sometimes lacking (especi-ally in patients with localized aggressive periodontitis)	Commensurate with the amount of etiological factors present

According to the patient's age EOP is divided into:

– prepubertal (younger than 11 years);
– juvenile (12–21 years);
– rapidly progressing (21–35 years).

According to the form EOP is divided into:

– localized;
– generalized.

The leading role belongs to the specific microflora (Gram-negative an-aerobes): Actinobacillus actinomycetemcomitans; Porphiromonas gindivalis; Prevotella(Bacteroides) intermedius; Bacteroides gingivalis; Bacteroides zoogle-formens; Capnocytophaga sputigena.

Phase microscopy has revealed the following in patients with EOP:

– the number of cocci and immobile rods is reduced by 1.5-2 times;
– the number of mobile forms of facultative anaerobes increases dramatically;

– the ratio of fixed and mobile forms of microorganisms is reduced by more than 3 times compared with the norm.

Bacterial colonization starts the process of periodontal destruction, but the result of this process depends on the protective forces and reactance of an organism that can both hinder and facilitate the destructive processes. ***Aggressive periodontitis almost doesn't depend on the level of oral hygiene, but largely depends on the immune status of patients.***

Autoimmune components of EOP development are the following:

– Functional defects of polymorphonuclear leukocytes and / or monocytes.

– Reduction of immunoglobulins adhesion and the formation of immune complexes lead to a prolonged duration of the inflammatory process.

– Sensitization of T lymphocyte is increasing.

– Ratio of the amount of T cells changes. The activation of polyclonal B-lymphocytes causes the production of antibodies, the release of inflammatory mediators and factors which activate the osteoclasts. The osteoclasts in their turn resorb the bone.

– As a result, autoaggression mechanisms are formed and lead to the progressive course of periodontitis destructive processes in the alveolar bone.

– Aggressive periodontitis is characterized by genetic changes in orientation of cellular immunity and autoimmune nature.

Prepubertal periodontitis

It occurs in children younger 11 years.

Features of the ***localized prepubertal periodontitis*** are the following:

– one or more teeth are affected;

– a slight gum inflammation;

– rather slow bone tissue destruction;

– specific microbial flora is represented by Capnocytophaga sputigena, Capnocytophaga gingivalis and Bacteroides gingivalis;

– Actinobacillus actinomycetemcomitans is not found (unlike juvenile periodontitis!!!).

Features of the ***generalized prepubertal periodontitis*** are the following:

– a very rare condition;

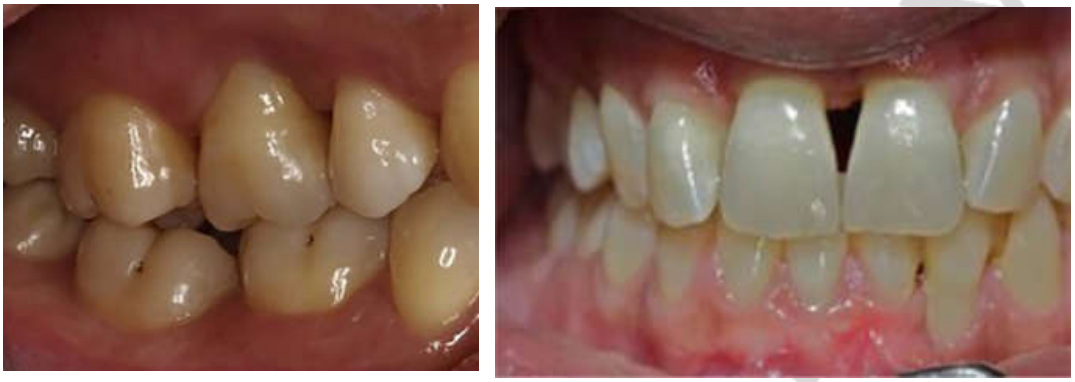
– periodontal tissues are inflamed;

– the rate of progressive destruction of the alveolar bone is high;

– the teeth become mobile and fall out very quickly.

It commonly begins at about 11–13 years during or shortly after the permanent eruption of the teeth. The pathology affects girls more frequently.

Localized juvenile periodontitis. Permanent molars and incisors are the first to become involved in the process (fig. 14). The process development in the periodontium occurs painlessly and rapidly.



a ***b***

Fig. 14. Localized aggressive periodontitis in an 18-year-old Caucasian, female, nonsmoking patient. Familiar aggregation was reported. (Dr. Hendrik Schulze, Bonn, Germany.):
a — clinical view showing recessions at the first molars; *b* — clinical view showing recessions at the incisors

There is mobility of teeth without visible gingival inflammation and periodontal pockets. The pockets appear later, usually in the region of the first upper molars, on one or more surfaces of the affected tooth. During the initial stage of the disease calculus is not typical; gum in the affected teeth may have a normal texture and color or a slight inflammation.

The degree of periodontal tissue destruction does not correspond to the local irritants (fig. 15).

X-ray diagnosis is necessary to confirm the diagnosis. Vertical resorption of the alveolar bone in the area of the first molars and incisors in apparently healthy adolescents is classic diagnostic feature of juvenile periodontitis.

X-ray examination in the early stages of the disease reveals damage only on one approximal surface. Next stages involve other approximal surfaces. X-ray signs include arcuate decline of alveolar bone (from the distal surface of the 2nd premolar to the medial surface of the 2nd molar). It is called «mirror-image» (fig. 16).

Generalized juvenile periodontitis. Resorption pockets are formed in the other teeth. There are signs of tooth migration, especially in the frontal area. The disease can have an acute onset, accompanied by fever. Redness, swelling, pain are marked. Inflammation quickly spreads to adjacent areas leading to the formation of periodontal pockets, tooth mobility, bone resorption, loss of teeth.

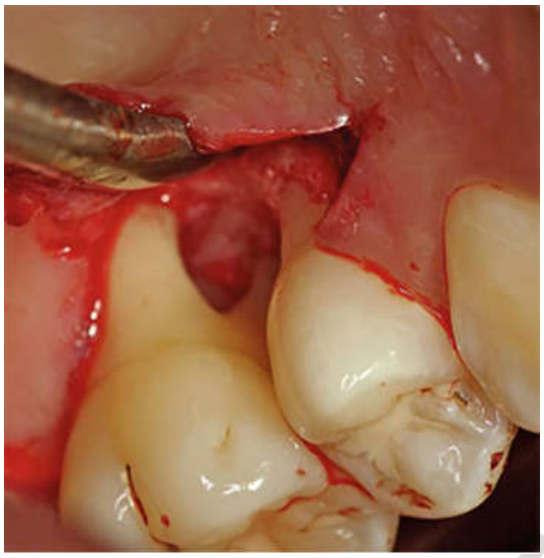


Fig. 15. Localized aggressive periodontitis in an 18-year-old Caucasian, female, nonsmoking patient. Intraoperative view showing advanced, localized, angular bony defect associated with a first molar (Dr. Hendrik Schulze, Bonn, Germany.)

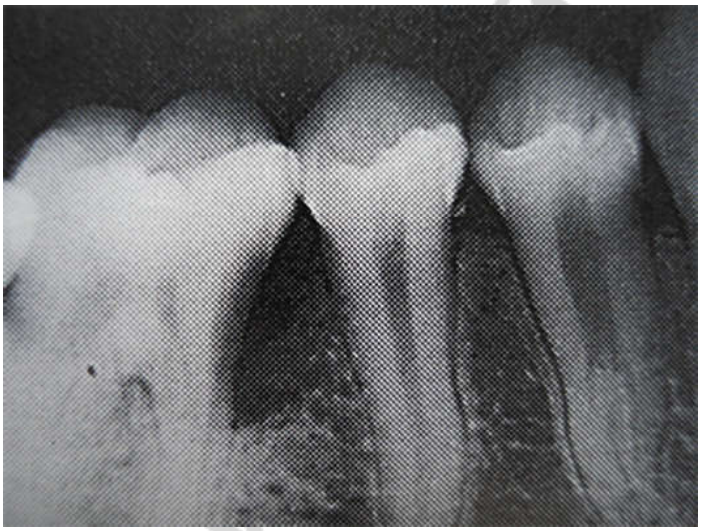


Fig. 16. «Mirror-image» on X-ray

Most common groups of microorganisms in dental plaque in patients with juvenile periodontitis are the following: Actinobacillus actinomycetemcomitans (95–100 %); Capnocytophaga sputigena; Bacteroides intermedius.

In response to microbial invasion the immune system produces antibodies. But the antibodies are not efficient and their amount in saliva and gingival fluid are reduced. This leads to rapid generalization of the process.

Generalized juvenile periodontitis is more common in people of Caucasian nationality, Arabs, Tatars, as well as in adolescents from mixed marriages. Blood group III may be a genetic marker.

Rapidly progressing periodontitis

It can develop in patients who have had juvenile periodontitis or in previously healthy individuals at the age of 21–35 years.

Rapidly progressing periodontitis has the same nature as juvenile periodontitis and similar microbial flora (Porphiromonas gingivalis is added).

Clinical features of a rapidly progressing periodontitis are the following:

- frequent exacerbations;
- short periods of remission;
- can be accompanied by fever, weight loss, depression.

Treatment of aggressive periodontitis

Treatment of this disease is complex. The main factor of a successful treatment is the inhibition of specific microorganisms. Prevalence of Actinobacillus actinomycetemcomitans is a sign of an active process. If disappearance or reduction of Actinobacillus actinomycetemcomitans amount is not achieved the disease persists or recurs.

There are two approaches in aggressive periodontitis treatment: perfect oral hygiene and adequate antibiotic therapy.

Perfect oral hygiene includes meticulous home oral care and professional oral hygiene.

Antimicrobial therapy should be followed by monitoring subgingival Actinobacillus actinomycetemcomitans and other potentially pathogenic bacteria. 3 months after completion of antimicrobial therapy microbiological monitoring is conducted for the assessment of treatment effectiveness.

Periodontal surgery and prosthodontic/orthodontic treatment should be used when they are required. All necessary manipulations which can not be performed by one specialist require help of dentists of all profiles (surgeon, therapist, orthodontist, and orthopedist).

Prognosis of aggressive periodontitis is unfavorable but early diagnosis and timely complex treatment allows stopping the rapid processes and achieving long period of remission.

PERIODONTITIS AS A MANIFESTATION OF SYSTEMIC DISEASE

In children with systemic diseases changes in periodontal tissues often develop. It is called symptomatic periodontitis. Clinical course and prognosis of symptomatic periodontitis depend on systemic disease course. Process stabilization occurs if the patient undergoes systematic treatment at the dentist and related

professionals; process progression occurs if the patient undergoes untimely and irrational treatment in decompensated states and genetically caused diseases.

Periodontitis as a manifestation of diabetes mellitus

Diabetes mellitus is a state of chronic hyperglycemia due to the action of many exogenous and endogenous factors. There are 2 types of diabetes mellitus : type 1 is more common in children and adolescents, whereas type 2 affects adults.

Pathogenesis of periodontal pathology is the following:

1. Insulin-dependent tissues do not receive glucose ─ the main source of energy.

2. In hyperglycemia hemoglobin is joined with glucose and loses its ability to transport oxygen to tissue; as a result hypoxia occurs.

3. Disrupted transport of amino acids into cells, protein synthesis, reduced reparative ability of tissues, reduced periodontal tissue resistance to the action of local factors and microbes.

4. Blood circulation is disturbed (microangiopathy occurs).

5. Diabetic neuropathy develops, the regulation of vascular tonus changes, etc.

In patients with periodontitis as a manifestation of diabetes mellitus periodontal pockets with purulent bloody contents and succulant granulations are detected. Significant abnormal mobility of teeth and large amount of dental plaque are also noted. Teeth are moved, their rotations on the axis, and secondary position anomalies are observed.

X-ray findings are the following:

– disturbance of the cortical bone of the alveolar edge;
– osteoporosis of the interdental septa peaks;
– periodontal ligament is extended;
– diffuse osteoporosis;
– in severe forms of diabetes vertical resorption of interdental septa develops, deep bone pockets are formed. There are foci of osteolysis of the jaw body;
– features are: «corroded» edges resorption, deep bone pockets (down to the tooth apex); «cupped» resorption.

The treatment should be complex including endocrinological treatment. Total therapy aims are to achieve compensation through the appointment of special diet, insulin, cholesterol-lowering agents and lipotropic vitamins, hepatic, anabolic drugs. Local treatment includes professional hygiene, 0.06–0.12–0.2 % solution of chlorhexidine, herbal extracts for the mouth trays, physiotherapy (electrophoresis of 3–5% solution komplamin), curettage, splinting. In case of enamel caries in pits and fissures invasive sealing and preventive restoration should be used.

Periodontitis as a manifestation of hereditary neutropenia

Hereditary neutropenia is a group of rare inherited diseases with almost complete absence of neutrophils in the blood that can be detected continuously (constant neutropenia), or at regular intervals (periodic neutropenia).

Pustular lesions of the skin and mucous membranes, abscesses, stomatitis, blepharitis, suppurative processes in the lungs are common clinical signs of the hereditary neutropenia.

Generalized periodontitis is manifested during teething and is accompanied by ulcerative gingivitis. By the age of 3 years there are deep periodontal pockets, destruction of alveolar bone, primary teeth mobility. A similar picture is observed in the permanent dentition. The process progresses and results in a complete loss of teeth by 12–14 years (fig.17).

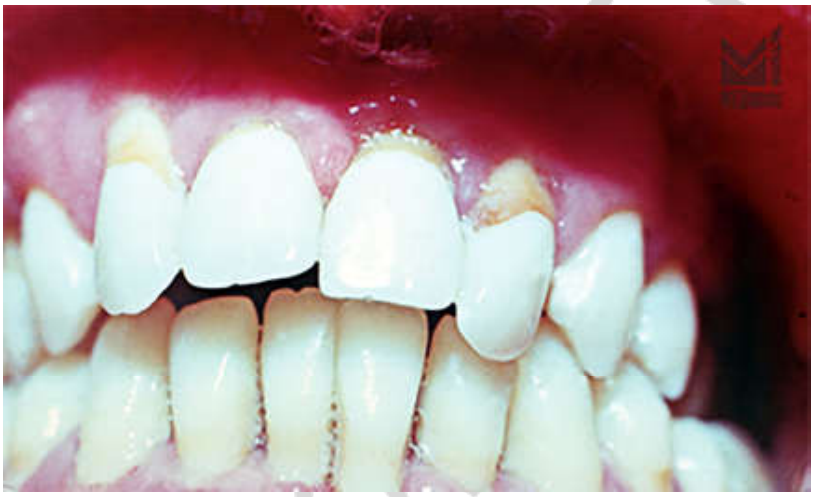


Fig. 17. Periodontitis as a manifestation of hereditary neutropenia. Clinical vew

On X-ray we can find destructive process in the bone of the jaw. It is limited to the area of the alveolar process and has clear boundaries.

Treatment should be complex including the hematologist. Periodontal treatment is symptomatic. Movable teeth are removed during remission.

Periodontitis as a manifestation of histiocytosis X

Histiocytosis X is a generic term that refers to the increase of histiocytes, type of white blood cell that act as scavengers to remove foreign material from the blood and tissues. Since recent research has demonstrated Langerhan cell involvement as well as histiocytes, the term Langerhans Cell Histiocytosis (LCH) can be used instead of histiocytosis X.

Either term refers to three separate illnesses (listed in order of increasing severity): eosinophilic granuloma, Hand-Schuller-Christian disease and Letterer-Siwe disease. Now according to the course of the disease and the degree of in-

volvement in the pathological process of different tissues and organs Langerhans cells histiocytosis are divided into four main clinical forms:

– I — disturbance of one of the bones of the skeleton;
– II — generalized disturbance of the bone system;
– III — generalized disturbance of the bone and lymphatic systems;
– IV — generalized disturbance of the bone and lymphatic systems combined with visceral manifestations.

Etiology is not clear. *Pathogenesis* is the formation of granulomas. Accumulation of Langerhans cells is formed in the bone tissue. Then macrophages come in the focus, bone tissue is destroyed and replaced by a granuloma. Sometimes lesions are rare and do not influence the health greatly, may disappear spontaneously. In other cases, granulomas form in many organs and change their function and may cause death.

Histological changes are permanent, and do not depend on the severity of the clinical picture. The main feature of the disease is pathological Langerhans cells, the markers are Birbek bodies, antigen SD1a and S-100 protein.

Taratynov disease (eosinophilic granuloma) = I form of histiocytosis X. The process is chronic, destructive changes are seen in one of the bones of the skeleton. In early stage of the disease oral manifestations are the first and the only symptom of the disease. When the process is localized in the mandible clinical features are:

– discomfort in the mouth,
– rapidly increasing mobility,
– loss of teeth,
– gum recession or ulceration.

When teeth are removed the alveoli are filled with granulations, which do not heal for a long time. On X-ray picture the dentist finds rounded transparent area with marked sclerosis zone on the periphery and wave-shaped boundaries. The roots of the teeth are «hanging in the air».

Hand-Schuller-Christian disease = II-III form of histiocytosis X. The disease develops slowly, with periods of remission for many years. Generalized bone lesion is typical. *Clinical picture* of the acute period of the disease is characterized by the following:

1) diabetes mellitus (increased thirst, polyuria, hepatosplenomegaly, etc .);

2) exophthalmos ─ protrusion of the eyeball due to granulomatous growth in retro-bulbar direction of the orbit;

3) periodontitis.

Periodontitis is characterized by:

– ulcerative gingivitis,
– putrid breath,
– tooth mobility,
– teeth roots are exposed and covered with plaque,

– deep periodontal pockets with ulcerated edges and granulation but without suppuration.

X-ray findings include generalized bone disturbances and lacunar resorption. Alveolar bone resorption is combined with the centers of destruction in other parts of the jaw, which are not associated with the teeth. Other flat bones are also affected: skull, sternum, pelvis, shoulder blades. Structureless defects of different sizes with jagged, but precisely outlined contours are defined in the area of the parietal, temporal and frontal bones

Letterer-Siwe disease = IV form of histiocytosis X. The onset of the disease is acute: fever, disturbance of sleep and appetite, lethargy, drowsiness, loss of body weight may be observed. Often these *clinical symptoms* are perceived as common childhood illnesses. Later a maculopapular rash may occur on the scalp, behind the ears, on the chest. Heavy lymphadenitis, otitis, stomatitis, diabetes insipidus, thirst, polyuria, enlargement of the spleen and liver, diarrhea, symptoms of secondary diseases of the cardiovascular system, exophthalmos join to these symptoms. Generalized periodontitis with progressive osteolysis develops.

X-ray findings include osteoporosis, diffusive resorption of interdental septi and focal resorbtion of the jaw body.

Prognosis is unfavorable.

The treatment of histiocitosis X depends on the course of the disease, defect localization and the patient's age. Treatment is carried out at the Department of Oncology and Hematology. If the patient has a localized lesion, bone scraping with local medical therapy (corticosteroid) is possible. If the patient has generalized lesions, chemotherapy (cytostatics) and radiotherapy combined with immunocorrection are possibly used. Dental care is symptomatic.

Periodontitis as a manifestation of hypoimmunoglobulinemia

Hypoimmunoglobulinemia is a disease that is characterized by a deficiency of antibodies and (as a result) increased susceptibility to bacterial infections.

Clinical picture is characterized by the development of abscesses, boils, ear infections, pneumonia, pyelonephritis, arthritis, enterocolitis, pyoderma, etc. Mild reaction of the lymph nodes, as well as immunity to viral aggression, are typical. In the oral cavity: chronic hypertrophic gingivitis with the growth of the gingival margin, which covers the entire tooth crown, is typical. There are deep pathological gingival pockets, but no dental calculus in this pathology.

Destruction of the alveolar bone tissue is found on X-ray.

Diagnosis includes the study of serum immunoglobulins.

Dental *care* is symptomatic.

Periodontitis as a manifestation of Papillon-Lefevre syndrome

Papillon-Lefevre syndrome is a hereditary (autosomal recessive) disease. It was described in 1924.

The *main feature* of the syndrome is a combination of palmar-plantar dyskeratosis and periodontitis (fig. 18). Dyskeratosis (in the form of alternating areas of hyperkeratosis and increased exfoliating of the epidermis) affects the palms, soles, and sometimes the knee and elbow joints. The main symptoms of the disease are manifested at birth (dyskeratosis) and with the first teeth eruption (periodontitis).

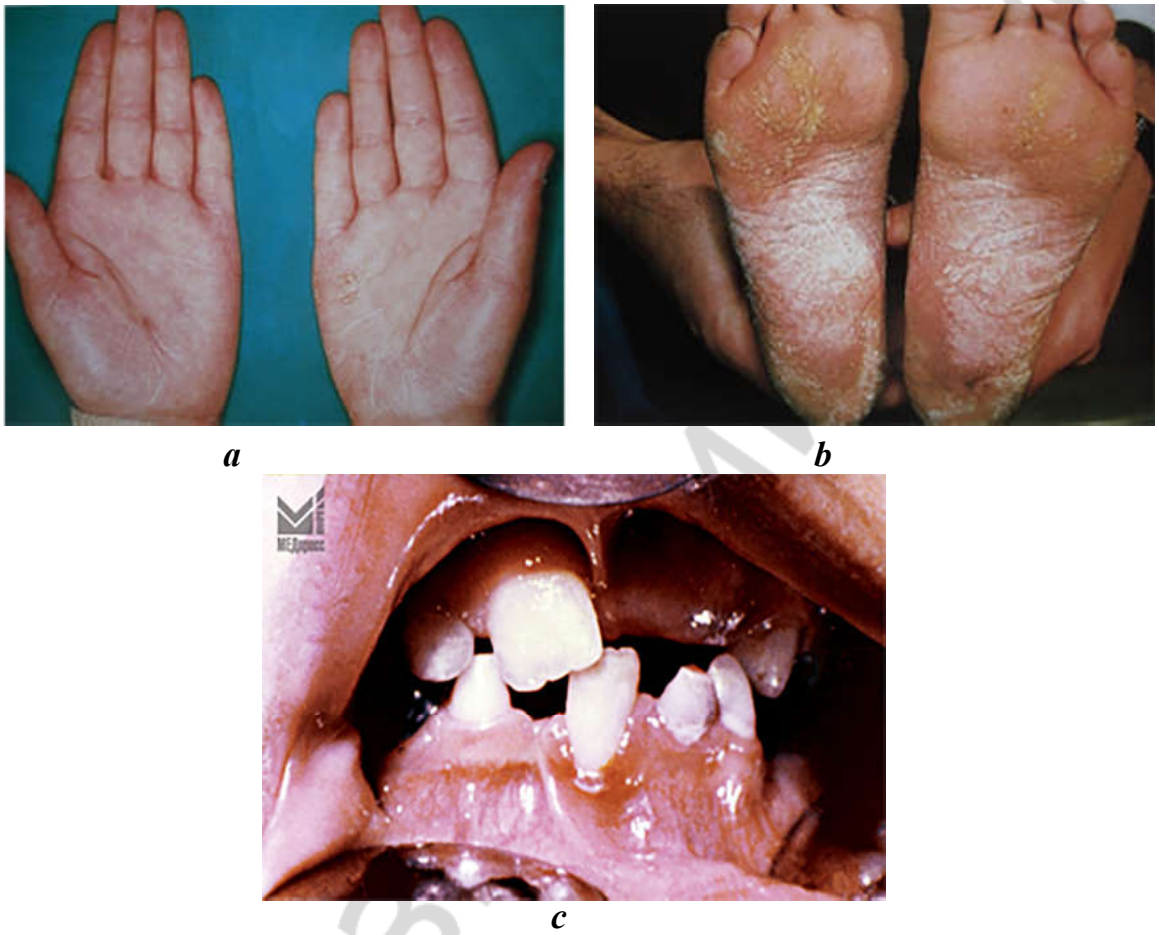


Fig. 18. Periodontitis as a manifestation of hereditary neutropenia. Clinical vew: *a* — hands; *b* — foots, *c* — oral manifestations

Gums around the temporary teeth are swollen, hyperemic and bleeding, deep periodontal pockets with purulent exudate, abscesses are observed. At the age of 4–5 years old children almost have no temporary teeth, gums become smooth and shiny. However with the eruption of permanent teeth prior symptoms are detected again. Permanent teeth eruption is accompanied by the destruction of the bone tissue and alveolar bone dystrophy. Alveolar bone defects are clearly defined and form wave-shaped outline on X-ray. Complete tooth loss occurs at about 14–15 years.

General condition of the child is manageable. Disturbance of tryptophan metabolism is possible.

The treatment should be complex in cooperation with the pediatrician and dermatologist. Cutaneous manifestations of the disease are treated topically with keratolytic pastes and ointments. Systemic therapy with retinoids is also prescribed. The use of drugs which normalize pathological tryptophan metabolism in combination with local anti-inflammatory treatment leads to some improvement in the local oral status. At the age of 4 years and older prosthetic treatment can be imlemented.

Professional oral hygiene with application of 0.12–0.2% solution of chlorhexidine and gel «Metrogil-dent» should be done. Maintenance therapy should be carried out 4 times a year with the use of mouth rinses, toothpastes and gels based on chlorhexidine.

The possibility and practicability of antibiotic therapy and using implants are being studied.

Periodontitis as a manifestation of hypophosphatasia

Hypophosphatasia is a hereditary disease transmitted in an autosomal recessive manner. Serum alkaline phosphatase levels decrease and phosphoethanolamine is detected in the urine. It leads to defective mineralization of bone and/or teeth.

There are at least six clinical forms of the pathology based on the age of diagnosis and severity of features. Clinical manifestations range from stillbirth without mineralized bone in case of severe outcome to pathologic fractures of the lower extremities in later adulthood in case of mild outcome.

Hypophosphatasia leads to severe dental caries due to insufficient mineralization of the enamel and dentin. Teeth mobility and premature exfoliation of primary teeth occur due to cementogenesis disturbance. Cement is hypoplastic or absent, connective tissue between the tooth and the bone is rapidly destroyed, bone is resorbed. As a result primary teeth are lost too early.

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CONTENT

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Шаковец Наталья Вячеславовна
Бурак Жанна Михайловна
Кленовская Маргарита Игоревна

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