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GINGIVITIS.
ETIOLOGY. DIAGNOSIS. TREATMENT.

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1 THE TISSUES OF THE PERIODONTIUM

The periodontium is the complex of the tooth supporting tissues (Greek: peri- „around"; odont- „tooth"), united by the common function and origin and consists of:

– the gingiva
– the alveolar bone
– the periodontal ligament
– the cementum

1.1 Structure of the gums

Three parts of the gums are distinguished:

– the marginal gingiva
– the attached gingiva
– the interdental gingiva

The marginal gingiva (unattached gingiva)

It consists of a central core of connective tissue covered by stratified squamous epithelium.

It is demarcated from the adjacent attached gingiva by a shallow linear groove. It may be separated from the tooth surface with a periodontal probe.

The epithelium along the inner surface (facing the tooth) is neither keratinized nor parakeratinized and forms the lining of the gingival sulcus.

The marginal gingiva in normal periodontal tissues extends approximately 2 mm coronal to the cemento-enamel junction (CEJ). The space between the marginal gingiva and the external tooth surface is termed the gingival sulcus. The normal depth of the gingival sulcus, and corresponding width of the marginal gingiva, is variable. In general, sulcus depths

less than 2mm to 3mm in humans and animals are considered normal (Manfra-Maretta S.,1990).

The interdental gingiva occupies the gingival embrasure. It consists of two papillae and the col.

The attached gingiva is firm and tightly bound to the periosteum of alveolar bone.The facial aspect of the attached gingiva extends to the relatively loose and movable alveolar mucosa from which it is demarcated by the mucogingival junction.

The epithelium is differentiated into:

1) a cuboidal or columnar basal layer;

2) a spinous layer, comprised of polygonal cells;

3) a multilayered granular component consisting of flattened cells with prominent basophilic keratohyaline granules;

4) a cornified layer.

The epithelium is joined to the underlying connective tissue by a basal lamina. It is synthesized by the basal epithelial cells and consists of a polysaccharide-protein complex and collagen (reticulin) fibers.

It is permeable to fluids but acts as a barrier to particular matter.

The gingival fibers

The connective tissue of the marginal gingiva is densely collagenous, containing a prominent system of collagen fiber bundles:

Their functions are:

- to brace the marginal gingiva firmly against the tooth
- to provide the rigidity necessary to withstand the forces of mastication without being deflected away from the tooth surface
- to unite the free marginal gingiva with the cementum of the root and the adjacent gingiva.

3 groups of gingival fibers are known: gingivodental, circular, transseptal.

Connective tissue cellular elements:

– ***fibroblasts*** synthesize and secrete the collagen fibers as well as elastin and non-collagenous proteins, glucoproteins and glucosaminoglycans;

– ***mast cells*** contain a variety of biologically active substances, such as histamine, proteolytic enzymes „slow-reacting substances", heparin; all of them may be involved in the development and progress of gingival inflammation.

Small amount of ***plasma*** *cells* and ***lymphocytes*** are almost always found in the connective tissue.

Gingival ***plasma cells*** are numerous in the lamina propria in the vicinity of blood vessels. These cells produce antibodies (i.e., IgG, IgA, or IgM) directed against local antigen.

Neutrophils can be seen in relatively high numbers in both the gingival connective tissue and the sulcus. It is common to see them migrating through the sulcular and junctional epithelium. These cells perform a protective role by phagocytizing bacteria and other foreign substances. They contain lysosomes which in turn contain a variety of hydrolytic enzymes that kill bacteria after phagocytosis. When neutrophils die these enzymes are released and may contribute to tissue destruction.

Macrophages – large phagocytic cells are also numerous in the gingival lamina propria.

The gingival sulcus, sulcus epithelium and junctional epithelium

The marginal gingiva forms the soft tissue wall of the gingival sulcus, and is joined to the tooth at the base of the sulcus by the functional epithelium. The sulcus is lined with thin, non-keratinized stratified epithelium without rete pegs. It

is extremely important, because it may act as a semipermeable membrane through which injurious bacterial products pass into the gingiva and tissue fluid from the gingiva seeps into the sulcus.

The epithelial attachment of the junctional epithelium consists of a basal lamina that is comparable to that which attaches epithelium to the connective tissue elsewhere in the body.

The basal lamina consists of a lamina densa (adjacent to the enamel) and a lamina lucida to which hemidesmosomes are attached. Organic strands from the enamel appear to extend into the lamina densa. The junctional epithelium attaches to afibrillar cementum when it is present.

The attachment of the junctional epithelium to the tooth is reinforced by the gingival fibers, which brace the marginal gingiva against the tooth surface. For this reason the junctional epithelium and the gingival fibers are considered a functional unit, referred to as the dentogingival unit.

Questions

1. What are the constituent parts of the gums?
2. What is the histological structure of the gingival tissues?
3. What types of epithelium can be distinguished in the gums?
4. Comment on the structure of connective tissue in the gums?
5. What is referred to as dentogingival junction?

1.2. Structure of the Alveolar Bone

Normal microscopic features

The ***alveolar process*** is the bone which forms and supports the tooth sockets (alveoli). It consists of the inner

socket wall of thin compact bone called the ***alveolar bone proper (cribriform plate)***, the ***supporting alveolar*** bone which consists of cancellous trabeculae and the facial and lingual plates of a compact bone. The interdental septum consists of cancellous supporting bone enclosed within a compact border.

The alveolar process is divisible into separate areas on an anatomic basis, *but it functions as a unit. All parts are interrelated in the support of the teeth.* Occlusal forces which are transmitted from the periodontal ligament to the inner wall of the alveolus are supported by the cancellous trabeculae, which in turn are buttressed by the labial and lingual cortical plates. Designation of the entire alveolar process as the alveolar bone is more consistent with its functional unity.

Cells and Intercellular Matrix

Alveolar bone is formed during fetal growth by intramembranous ossification, and consists of a calcified matrix with osteocytes enclosed within spaces called lacunae. The osteocytes extend processes into canaliculi which radiate from the lacunae. The canaliculi form an anastomosing system through the intercellular matrix an important role in the mineralization process. Osteoblasts produce this material together with other matrix constituents. A similar series of events is believed to occur during dentin matrix production and mineralization.

Osteoclasts are large, multinucleated cells that are often seen on the surface of bone within eroded bony depressions referred to as Howship's lacunae. The main function of these cells is considered to be resorption of bone. When they are active, as opposed, to resting, they possess an elaborately developed ruffled border from which hydrolytic enzymes are believed to be secreted. These enzymes digest the organic portion of bone. The activity of osteoclasts and the morphology of the ruffled border can be modified and regulated by

hormones such as parahormone and calcitonin. The origin of osteoclasts is still a matter of speculation and controversy.

The principal fibers of the periodontal ligament which anchor the tooth in the socket are embedded for a considerable distance into the alveolar bone where they are referred to as ***Sharpey's fibers***. Some Sharpey's fibers are completely calcified. The socket wall consists of dense lamellated bone, some of which is arranged in Haversian system and "bundle bone". Bundle bone is the term given to a bone adjacent to the periodontal ligament because of its content of Sharpey's fibers.

The ***cancellous portion*** of the alveolar bone consists of trabecular which enclose irregularly shaped marrow spaces. There is a wide variation in the trabecular pattern of the cancellous bone, which is effected by occlusal forces. The cribriform plate of the tooth socket appears radiographically as a thin, radiopaque line, termed the lamina dura. In the embryo and newborn, the cavities of the cancellous bones are occupied by the red hematomarrow. The red marrow gradually undergoes a physiologic change to the fatty or yellow inactive type of marrow.

The vascular supply of the bone is derived from blood vessels branding off of the superior or inferior alveolar arteries. Blood vessels branch extensively and travel through the periosteum. The endosteum lies adjacent to the marrow vasculature. Bone growth occurs by apposition of an organic matrix that is deposited by osteoblasts.

Bone is composed principally of the minerals calcium and phosphate, along with hydroxyl, carbonates, citrate, and trace amounts of other ions such as sodium, magnesium, and fluorine. The mineral salts are in the form of hydroxyapatite crystals of ultramicroscopic size and constitute approximately 65 to 70 per cent of the bone structure. The organic matrix consists mainly (90 per cent) of collagen (type I), with small amounts of noncollagenous proteins, glycoproteins, phospho-

proteins, lipids, and proteoglycans. The apatite crystals are generally aligned with their long axes parallel to the long axes of collagen fibers, and appear to be deposited upon and within the collagen fibers. In this fashion bone matrix is able to withstand heavy mechanical stresses applied to it during function.

Although the alveolar bone tissue is constantly changing in its internal organization, it retains approximately the same form from childhood through adult life. Bone deposition by osteoblasts is balanced by resorption by osteoclasts during the processes of tissue remodelling and renewal.

The bone matrix that is laid down by osteoblasts is not mineralized and is referred to as a prebone or osteoid. While new prebone is being deposited, the older prebone located below the surface becomes mineralized as the mineralization front advances. Recent investigations have revealed the morphologic and biochemical steps that are involved during the elaboration of bone matrix collagen. Briefly, procollagen molecules are synthesized and assembled by the rough endoplasmic reticulum and Golgi apparatus respectively within osteoblasts. Secretory granules then carry the procollagen aggregates to the cell surface for secretion to take place. At some time prior or subsequent to discharge from the cell, the procollagen molecules interact with a peptidase and become converted to tropocollagen molecules which then have the capacity to assemble into typical collagen fibrils.

Prior to becoming mineralized, bone matrix collagen becomes coated or associated with a glycoprotein (or proteoglycan) material which, in the electron microscope, appears as an opaque granular substance.

Age changes in the periodontium

In the gingiva the following changes have been identified with ageing: recession, diminished keratinization, increased width of attached gingiva, decreased connective tissue cellularity, an increase of intercellular substances, loss of elasticity, a reduced oxygen consumption, a measure of metabolic activity.

In the periodontal ligament, ageing results in an increase in elastic fibers, decrease in vascularity, mitotic activity. Decreased width of periodontal slot which may result from encroachment upon the ligament by continuous deposition of the cementum and a bone. In alveolar bone in addition to reduction in height (senile atrophy), osteoporosis, decreased vascularity, and a reduction in metabolism and healing capacity occurs.

The most obvious change in the teeth with ageing is a loss of tooth substance caused by attrition.

Questions

1. Where are the compact and cancellous bones located in the alveolar process?
2. Ennumerate the main cells in the bone? Comment on their function?
3. What is the biochemical structure of the bone?
4. What age changes occurs in the periodontium?

1.3. The Periodontal Ligament

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 the 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 (acid-resistant) fibers, which are distributed mainly around the blood vessels and embedded in the 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 termed the "epithelial rests of Malasser".

Periodontal ligament fibroblasts synthesize collagen and they have been shown to possess the capacity to phagocytose in „old" collagen fibers and degrade them by enzyme hydrolysis.

Epithelial rests proliferate when stimulated and participate in the formation of periapical cysts and lateral root cysts.

The periodontal ligament may also contain calcified masses called cementicles.

The physical function of the periodontal ligament entails the following: transmission of occlusal forces to the bone; attachment of the teeth to the bone; maintenance of

gingival tissues in their proper relationship to the teeth; resistance to the impact of occlusal forces; protection of the vessels and nerves from the injury by mechanical forces.

Questions

1. Give the definition of the periodontal ligament
2. What groups of fibers according to their attachment are distinguished in the periodontal ligament?
3. What types of fibers are there in the periodontal ligament?
4. What are the cell elements of the periodontal ligament?
5. Enumerate the main functions of the periodontal ligament.

1.4. The Cementum

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

There are two main forms of the 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 the cementum: Sharpey's and the fibers belonging to the cementum matrix per se produced by the cementoblasts.

The inorganic content of cementum (hydroxyapatite, Ca10(PO4)6(OH)2) is 45-50 per cent, which is less than that of bone (65 per cent), enamel (97 per cent) or dentin (70 per cent).

Histochemical studies indicate that the matrix of cementum contains a carbohydrate-protein complex. Neutral and acid mucopolysaccharides are present in the matrix and cytoplasm of some cementoblasts.

The thickness of cementum on the coronal half of the root varies from 16 to 60 microns, or about the thickness of a hair. It attains its greatest thickness of up to 150 to 200 microns in the apical third and also in the bifurcation and trifurcation areas. Between the ages of 11 and 70 the average thickness of the cementum increases three-fold, with the greatest increase in the apical region. Average thickness of 95 microns at the age 20 and 215 microns at age 60 have been reported.

Cementum deposition continues throughout the life.

Questions

1. What types of the cementum do you know?
2. Comment on the structure of the cellular cementum.
3. What is the biochemical structure of the cementum?
4. What is the average cementum thickness?

2. CURRENT CLASSIFICATIONS OF PERIODONTAL DISEASES

2.1. Classification by Danilevskij N.F., 1994

I. Inflammatory diseases.

1. Gingivitis. Papillitis (inflammation of interdental papillae).

Forms: catarrhal, hypertrophic, ulcerative, atrophic.

The course of the disease: acute, chronic.

The depth of the lesion: in the gingival tissues, osteoporosis of interdental septa.

The spreading of the pathological process: localized, generalized.

2. Localized periodontitis

Forms: catarrhal, hypertrophic, ulcerative, atrophic.

The course of the disease: acute, chronic.

The depth of the lesion: gingival tissues, alveolar bone.

The stage of the disease progression: initial stage, I stage, II stage, III stage.

The spreading of the pathological process: localized.

II. Dystrophic – inflammatory diseases.

1. Generalized periodontitis

The course of the disease: chronic, exacerbation, stabilization.

The stage of the disease progression: initial stage, I stage, II stage, III stage.

The spreading of the pathological process: diffuse process in the periodontal tissues.

2. Periodontosis (Periodontosis).

The course of the disease: chronic.

The stage of the disease progression: initial stage, I stage, II stage, III stage.

The spreading of the pathological process: diffuse injury of the periodontal tissues.

III. Progressing idiopathic diseases of the periodontium.

1. Periodontal diseases in the presence of hematologic diseases (cyclic neutropenias, leukemias, agranulocytosis).

2. Histiocytosis X: eosinophylic granuloma (Taratynov's diseases).

3. Periodontal diseases in the endocrinologic diseases (Niemann-Pick disease, Gaucher's disease, Papillon-Lefevre syndrome).

4. Periodontal condition in the hereditary diseases (Down syndrome, akatalasia, desmodontosis).

IV. Tumor-like processes in the periodontal tissue.

Benign tumors
Malignant tumors

2.2. Some others classifications of periodontal diseases

Page and Schroeder, 1982
Prepubertal periodontitis
Generalized
Localized
Juvenile periodontitis
Rapidly progressing periodontitis
Adult type periodontitis

Grant, Stern, and Listgarten, 1988
Bacterially induced diseases
Gingivitis
Periodontitis
Adult type
Postjuvenile
Early onset
Juvenile
Localized
Generalized
Acute necrotizing ulcerative gingivitis
Acute abscess
Pericoronitis
Functionally induced diseases
Traumatic occlusion
Disuse atrophy
Trauma
Habits, accidents

Suzuki, 1988

Adult periodontitis
Rapidly progressing periodontitis
Type A
Type B
Juvenile periodontitis
Postjuvenile periodontitis
Prepubertal periodontitis

World Workshop in Clinical Periodontics, 1989
Adult periodontitis
Early onset periodontitis
Prepubertal
Generalized or localized
Juvenile
Generalized or localized
Rapidly progressive periodontitis
Periodontitis associated with systemic diseases
Down syndrome
Diabetes type I
Papillon-Lefevre syndrome
AIDS
Other diseases
Necrotizing ulcerative periodontitis
Refractory periodontitis

Genco,1990
Periodontitis in adults
Periodontitis in juveniles
Localized form
Generalized form
Periodontitis with systemic involvement
Primary neutrophil disorders
Secondary or associated neutrophil impairment
Other systemic diseases; Miscellaneous conditions

Ranney, 1993

Gingivitis

Gingivitis, plaque bacterial

Nonaggravated

Systemically aggravated by sex hormones, drugs, systemic disease

Necrotizing ulcerative gingivitis

Systemic determinants unknown

Related to HIV

Gingivitis, nonplaque

Associated with skin disease; allergic; infectious

Periodontitis

Adult periodontitis

Nonaggravated

Systemically aggravated (neutropenias, leukemias, lazy leukocyte syndrome, AIDS, diabetes mellitus, Crohn's disease, Addison's disease)

Early-onset periodontitis

Localized early-onset periodontitis

Neutrophil abnormality

Generalized early-onset periodontitis

Neutrophil abnormality; immunodeficient

Early-onset periodontitis related to systemic disease

Leukocyte adhesion deficiency, hypophosphatasia, Papillon-Lefevre syndrome, neutropenias, leukemias, Chediak-Higashi syndrome, AIDS, diabetes mellitus type I, trisomy 21, histiocytosis X, Ehlers-Danlos syndrome (type VIII)

Early-onset periodontitis, systemic determinants unknown

Necrotizing ulcerative periodontitis

Systemic determinants unknown

Related to HIV

Related to nutrition

Periodontal abscess.

SOME SYSTEMIC DISEASES INVOLVING THE PERIODONTAL TISSUES

1.Necrotizing ulcerative periodontitis
   a. AIDS-associated
   b. Non-AIDS associated
2. Disorders of neutrophil function
   a. Agranulocytosis
   b. Cyclic neutropenia
   c. Chediak-Higashi syndrome
   d. Other diseases
3. Hematologic diseases
   a. Leukemia
   b. Anaemias
   c. Histiocytosis X
4. Metabolic diseases
   a. Gaucher's disease
   b. Niemann-Pick disease
5. Connective tissue disorders
   a. Ehlers-Danlos syndrome
   b. Wegener's granulomatosis
   c. Sarcoidosis
6. Bone diseases
   a. Hypophosphatasia
   b. Paget's disease
7. Neoplastic diseases
   a. Benign tumors
   b. Malignant tumors

INTERNATIONAL CLASSIFICATION OF THE DISEASES, 1998

All the necessary for Health services information is included in the international diseases classification with the series of modules, connected with the „family" classification conception. This classification is build in three and four steps levels of alphabet-figure scheme of coding, what gives the possibility to balance chapters contents and leave enough space for future changes.

Diseases of the oral cavity, salivary glands and jaws as a separate part K00-K14 are included into group of the Diseases of digestive system class XI part K00-K93. This class has 9 part more of digestive system diseases: based on anatomical classification.

The abbreviator (WFI) – without future instructions.

Diseases of the oral cavity, salivary glands and jaws (K00-K14)

K00	Anomalies of teeth development and eruption
K01	Retained and impacted teeth
K02	Dental caries
K03	Other diseases of teeth hard tissues
K04	Diseases of the pulp and periapical tissues
K05	Gingivitis and periodontal diseases
K06	Other diseases of the gums and edentulous jaws
K07	Oral-facial anomalies (including bite anomalies)
K08	Other injuries of teeth and bite anomalies
K09	Cysts of the oral cavity which are not classified in other parts
K10	Other diseases of the jaws
K11	Diseases of the salivary glands
K12	Stomatitis and injuries connected with them

K13 Other diseases of the lips and oral mucous membrane

K14 Diseases of the tongue

K00 Anomalies of teeth development and eruption
With the exception: retained and impact teeth (K01.-)

K00.0 Adentia
Hipodentia
Oligodentia

K00.1 Supercomplected teeth
Distomolares
The fourth molar
Mesiodentia
Paramolars
Additional teeth

K00.2 Anomalies of size and shape of the teeth
Growing together
Merging teeth
Derminating

Tooth:
* prominent
* invaginated
* tooth in the tooth

Enamel pearls
Macrodentia
Microdentia
Core teeth
Taurodontizm

Paramolar tubercular (cuspids)

With the exception: tubercular carabella anomaly, which is considered normal

K00.3 Pointed teeth

Teeth fluorosis
Pointed enamel
Nonfluorose darkening of the enamel

With the exception: teeth deposits (K03.6)

K00.4 Changes in teeth formation

Aplasia and hypoplasia of the tooth cement
Crack of the enamel
Enamel hypoplasia (neonatal, postnatal, prenatal)
Regional odontodysplasia
Turner's teeth

With the exception: Hetchinson's teeth and molar in born syphilis (A50.5)
Pointed teeth (K00.3)

K00.5 Inherited disturbance in teeth structure not included in other parts

Amelogenesis
Dentinogenesis uncompleted
Odontogenesis
Dentin dysplasia
Shell-teeth

K00.6 Disturbance in teeth eruption

Early eruption
The tooth erupted during the birth
The tooth of newborn prematurely erupted

Premature
* tooth eruption
* coming out of deciduous tooth
Preservation of deciduous tooth

К00.7 Syndrome of teeth eruption

К00.8 Other disturbances in teeth development
Changing of tooth color during its formation
Prominent teeth coloring

К00.9 Disturbance in tooth development
Disturbance in the odontogenesis

К01 Retained and impacted teeth
With the exception: retained and impacted teeth with anomalies in their position or the position of neighboring teeth (К07.3)

К01.0 Retained teeth
Retained tooth – the tooth which changed its position during the eruption without the obstruction with another tooth

К01.1 Impacted tooth
Impacted tooth – tooth which has changed its position during the eruption because of the obstruction with another tooth

К02 Tooth caries

К02.0 Enamel caries
Lesion in the form of white spot (initial caries)

К02.1 Caries of tooth dentin

К02.2 Caries of tooth cement

К02.3 Stopped tooth caries

К02.4 Odontoclasia
Children melanoclasia
Melanodontoclasia

К02.8 Teeth caries of another type

К02.9 Teeth caries not defined precisely

К03 Other diseases of teeth hard tissues
With the exception: bruxism (F45.8)
dental caries (К02.-)
teeth grinding (F45.8)

К03.0 Exceeding teeth wear (attrition)
Attrition
* aproximal of the teeth
* occlusal

К03.1 Teeth grinding
Grinding:
* with a tooth powder
* usual
* professional of the teeth
* ritual
* traditional
Wedge-shaped defect

К03.2 Teeth erosion
Teeth erosion:

* Teeth grinding
* because:
  – diet
  – medications
  – constant vomiting
* idiopathic
* professional

K03.3 Pathological teeth resorption
Internal pulp granuloma
Resorption teeth hard tissues (external)

K03.4 Hypercementosis
Cement hyperplasia

K03.5 Teeth akylosis

K03.6 Dental deposits
Dental calculus:
* supragingival
* subgingival
Teeth deposits:
* betel induced
* black
* green
* white
* orange
* because of a cigarette smoking
Teeth coloration:
* WFI
* not corresponding with WFI

K03.7 Changing of the color in teeth hard tissue after their eruption

With the exception: teeth deposits (K03.6)

K03.8 Other defined diseases of teeth hard tissues
Irradiated enamel
Sensitive dentin
If the identification of the type of radiation is needed additional code of external reason is used (class XX)

K03.9 Uncertain disease of teeth hard tissues

K04 Diseases of the pulp and periapical tissues

K04.0 Pulpitis
Pulpal:
* abscess
* polylus
Pulpitis:
* acute
* chronic (hypertrophic) ulcerative
* purulent

K04.1 Pulp necrosis
Gangrene of the pulp

K04.2 Pulp degeneration
Denticles
Pulpal:
* calcinosis
* concrements

K04.3 Anomalous formation of hard tissues in the pulp
Secondary or irregular dentin

К04.4 Acute apical periodontitis of a pulp origin
Acute apical periodontitis (WFI)

К04.5 Chronic apical periodontitis
Apical or periapical granuloma
Apical periodontitis (WFI)

К04.6 Periapical abscess with a cavity
Dental abscess with a cavity
Dentoalveolar

К04.7 Periapical abscess without a cavity
Dental
Dentoalveolar abscess (WFI)
Periapical

К04.8 Root cyst
The cyst:
* apical (periodontal)
* periapical
* residual root cyst
With the exception: lateral periodontal cyst (К09.0)

К04.9 Other and uncertain diseases of pulp and periapical tissues

К05 Gingivitis and Periodontal diseases

К05.0 Acute gingivitis
With the exception: acute necrotizing ulcerative gingivitis (A69.1)
Herpetic gingivostomatitis (B00.2)

К05.1 Chronic gingivitis
Gingivitis (chronic):
* WFI
* Desquamative
* Hyperplastic
* Simple marginal
* Ulcerative

К05.2 Acute periodontitis
Acute periocorontitis
Periodontal abscess
With the exception: acute apical periodontitis (К04.4)
periapical abscess (К04.4)
* with a cavity (К04.6)

К05.3 Chronic periodontitis
Chronic pericoronitis
Periodontitis:
* WFI
* Complicated
* Simple

К05.4 Periodontosis
Juvenile periodontosis

К05.5 Other periodontal disease

К05.6 Periodontal disease without a precise definition

К06 Other diseases of gums and toothless alveolar process (К08.2)

With the exception: (atrophied) toothless alveolar process (K08.2)

gingivitis:
* WFI (K05.1)
* acute (K05.0)
* chronic (K05.1)

K06.0 Gingival recession

Gingival recession (generalized) (localized) (postinfectional) (postoperative)

K06.1 Gingival hypertrophy

Gingival fibromatosis

K06.2 Injuries of gums and toothless alveolar process, connected with trauma

Hypertrophy due to the irritation of toothless alveolar process (hypertrophy due to the prosthetics)

If necessary to indentify the reason with additional code of external reason (class XX)

K06.8 Other specified lesions of gums and toothless alveolar process

Fibrous epulis
Atrophied toothless alveolar process
Giantcell epulis
Peripheral giantcell granuloma
Piogenic gums granuloma

K06.9 Lesions of gums and toothless alveolar process not specified

3. THE EPIDEMIOLOGY OF GINGIVAL AND PERIODONTAL DISEASES

The purposes or objectives of epidemiology are to increase the understanding of the disease process, thereby leading to methods of control and prevention. In addition, it attempts to discover populations at high and low risk and to define the specific problem under investigation. One of the most valuable techniques employed in dental epidemiology is the epidemiologic index.

Epidemiologic indices attempt to quantitate clinical conditions on a graduated scale, thereby permitting and facilitating comparison with other populations examined by the same criteria and methods. Unlike the absolute or definitive diagnosis it is possible to make on an individual patient, an epidemiologic index (i.e., numerical value) will estimate only the relative prevalence or occurrence of the clinical condition. In general, indices are actually underestimates of the true clinical condition.

The criteria of a good epidemiologic index are that it must be easy to use, permit the examination of many people in a short period of time, define clinical conditions objectively, be highly reproducible in assessing a clinical condition when used by one or many examiners, be amenable to statistical analysis, and be strongly related numerically to the clinical stages of the specific disease under investigation.

INDICES USED TO STUDY PERIODONTAL PROBLEMS

Although there are many indices for recording and quantitating the many entities included under the term periodontal disease.

They are divided into those that evaluate:

a. The degree of inflammation of the gingival tissues.
b. The degree of periodontal destruction.
c. The amount of plaque accumulated.
d. The amount of calculus present.

3.1. Indices Used to Assess Gingival Inflammation

Bleeding index (I. Deneha, National Medical University of Lviv. Department of Therapeutic Dentistry).

Bleeding upon probing is one of the characteristic signs of inflammation and lowered resistance of the capillaries. Abnormal bleeding from the gingiva or other areas of the oral mucosa that is difficult to control is an important clinical sign suggesting a hematologic disorder. The expression of bleeding can testify to different forms of periodontal inflammation. Bleeding detects the level of inflammation in the periodontal tissues. Hemorrhagic tendencies occur when the normal hemostatic mechanism is disturbed.

The stage of hemorrhage in scores	The characteristic of bleeding upon probing of the periodontal pocket
0	absent
1	weak – appears at the end of probing (in 15-20 sec) as a narrow line of blood in the papilla-marginal regions of inspected tooth
2	Moderate – appears during probing as a small spot of blood filling the interdental spaces and the neck region of inspected tooth

3	strenuous - appears at the beginning or during probing as the large hemorrhage which quickly stops (in 5 -10 sec), turning into the overflowed blood stain overlaying inspected and adjacent papilla-marginal regions of gums and teeth
4	expressed bleeding appearing at the beginning of the probing as strongly pronounced and large hemorrhage, lasting at the same level for some time (15-30 sec) slowly decreasing and stopping. It looks like the blood is overlaying inspected and adjacent areas in the sextants.

Bleeding index (IH) is calculated as the sum of score of inspected units being divided into the number of units

Criteria for scoring of bleeding index
0,8-1,5 – small bleeding
1,5-2,3 – moderate bleeding
2,3-3 and more – heavy bleeding

P.M.A. index in Parma modification.

Originally the P.M.A. Index consisted of counting the number of gingival units affected with gingivitis. This approach was predicated on the belief that the number of units affected would convey the degree or severity of gingival inflammation. The facial surface of gingiva around a tooth was divided into three gingival scoring units: the mesial dental papilla (P), the gingival margin (M), and the attached gingiva (A). The presence or absence of inflammation on each gingival unit is recorded as 1, 2, 3.

$$\text{Index PMA} = \frac{\text{summary of all analized units}}{3 \times \text{number of teeth} \times 100\%}$$

Criteria for scoring index PMA
up to 25% – slight gingivitis
25-50% – moderate –"–
more than 50% – heavy gingivitis

Gingival Index (Silness-Loe)
is scored in the region of $\frac{6\ 1 \mid 4}{4 \mid 1\ 6}$ teeth

Criteria for estimation:
1 – slight inflammation (small changes in the colour)
2 – moderate inflammation (redness, oedema, hyperplasia)
3 – heavy inflammation (large redness, ulceration)

$$GJ = \frac{\text{summary of the all analized regions}}{6}$$

Criteria for scoring of gingival index
0,1-1,0 – slight gingivitis
1,1-2,0 – moderate –"–
2,1-3,0 – heavy –"–

3.2. Indices for the Evaluation of Periodontal Destruction

The degree of periodontal destruction can be evaluated by:

CPITN – Community Periodontal Index of Treatment needs

This index is proposed by WHO.

Dentition is divided into sexstants upper and lower molars and premolars and front teeth.

All teeth are examined in the sexstant and the maximal score achieved near one of the teeth is taken as the sexstant's score.

Score	Treatment needs
1 - bleeding on probing	improvement of the oral hygiene
2 – supra- and subgingival calculus; probing depth up to 3 mm	professional oral hygiene
3 - probing depth 4-5 mm	complex treatment including the periodontal surgery
4 - probing depth more than 6 mm	complex treatment including the periodontal surgery

Universal Periodontal Index Zubachyk V. (1995) (UPI)

The essence of the proposed index lies in the fact that the state of periodontium is determined by two criteria: reversible – the state of gums and irreversible one-the state of a bone tissue. The data were evaluated by a three-number mark which corresponded to the common notions of light, middle and severe forms of inflammatory or dystrophy processes course. Severity of gingivitis is determined by the symptoms of inflammation covering the surface of gums while the severity of periodontium and periorontitis – by the level of a bone resorption, the latter identified by x-ray or instrumentally according to the length of dental root. For this purpose the latter is conditionally divided into 3 parts beginning from the enamel-cement border up to the dental apex, thereby evaluating the level of a bone destruction relatively to those parts. Periodontal tissues are examined visually including the probing method by a special capitate probe.

To estimate UPI we propose to divide teeth rows into 6 segments which are limited by teeth 18-14(1), 13-23(2), 24-

28(3), 38-34(4), 33-43(5), 44-48(6), in brackets is the sextants number. Surrounding tissues in sections 17, 16(1), 11(2), 26, 27(3), 37, 36(4), 31(5), 46, 47(6) must be examined.

The state of gums and bone is assessed near the indicated teeth from four sides whereby the highest index of affection is taken into consideration. Results of examining this group of teeth show the state of periodontium on both jaws. When some of the indicated teeth is absent, the tissue near the next to the absent tooth falls under examination. If more, than a half of teeth are absent the sextant is not registered.

In children up to 15 years of age only the state of gums is taken into account. If an index tooth is absent on examination it may be replaced by a fully erupted incisor or a premolar.

Criteria of assessing and coding the periodontal state are as follows:

Gum status

1 – slight inflammation from any side of a tooth which covers papillary and partially a marginal part of gums (slight gingivitis),

2 – moderate inflammation embracing the marginal gums (moderate gingivitis),

3 – severe inflammation of interdental papillae, marginal and alveolar parts of gums (severe gingivitis).

To assess the state of periodontium with various pathological symptoms each examined sextant must be charactesized by a twofigure code in which the first figure indicates the severity of gingivitis and the inflamed surface of gums, the second figure showing the depth of periodontal bone affection and correspondingly the degree of the detected pathology. Before the code there is an ordinary number of the examined segment concretizing the place of pathological process. The twofigure number may be put in segments as it is usually done in determining the index of CPITN.

1 – reduction in a bone height within the upper tooth third (slight degree of pathology),

2 – alveolar margine reaches half a tooth (middle degree of pathological process),

3 – Height level of interdental septum is lower than half a tooth root (severe degree of pathology). Recording samples: the number 210 (^{10}) shows that in the second sextant there is light gingivitis without visible affections of other periodontal tissues, 533 ($_{33}$) – in the 5^{th} sextant there is a severe form of periodontitis with a bad symptomatic gingivitis, 402 ($_{02}$) – in the 4^{th} sextant the periodontal state following a complex treatment (remission stage), 401, 502, 601 ($_{01}$ $_{02}$ $_{01}$) – signs of periodontosis with a moderate degree of severity on a lower jaw in the front group of teeth while at sides a slight form is diagnosed.

A twofigure mode of coding favours differential diagnosing of acute and chronic course of periorontitis. The conclusion is grounded on peculiarities of acute inflammation course: bright color and tissue edema can be easily seen on a major part of gums which exceeds the level of interalveolar septums resorption. Unlike acute, chronic flabby pathological process is mainly focused in clinical pockets and cannot be diagnosed in the surface of mucous membrane of alveolar ridge due to thickness of mucosa and inexpressiveness of symptoms. Therefore codes 21 or 22 indicate a chronic inflammation while 33 and especially 32 and 31 show exacerbation of chronic periodontitis or probably its absceding form (31).

It's not expedient to make conclusions of sextants in an individual because a real clinical picture of the disease becomes opaque. General diagnosis of periodontal pathology is determined according to the state of the worst sextant. If the pathological process embraces only one sextant it's a sign of

localization; if two or more sextants are involved one can be sure of diffuse affection.

Numerous variants of combinations (16) in a twonumber coding of a periodontal tissue state and simplicity of a threemark assessment for the degree of its affection gives an opportunity to mathematically characterize the diversity of parodontal symptoms. The use of reversible and irreversible components of index contributes to a better illucidation of the pathological process dynamics.

Thus, the change of index UPI from 32 to 22 and 12 and finally to 02 indicates the normalization of the periodontal tissue state for account of effective treatment of periodontitis. Gradual reduction of the first index size due to elimination of symptomatic gingivitis is an evidence of it. Stability of the second code figure „2" in the given case can be explained by the absence of complete reparative osseous regeneration following the middle affection periodontitis. Therefore, during examination the particular attention must be paid to the second index which determines not only the diagnosis but remains the principal criterion of stability in periodontium. The latter is of great importance in an out-patient management of a patient. Recommended is the use of UPI in determining the diagnosis of periodontal diseases. E.g., catarrhal gingivitis 230 instead of chronic local catarrhal gingivitis, severe form; periodontitis 422, 523, 622 – chronic diffuse periodontitis, severe form; periodontosis 01 – mild form, in addition, figure expression indicates the localization of the pathological process. Approbation of UPI on 120 patients with various periodontal pathologies confirms the expediency of its application in practical medicine.

Coding simplicity and renewing of the clinical picture of disease make it possible to master quickly the methods and use UPI in epidemiological examination of population. According to the acquired data one calculates the indices of

proliferation (percent of individuals with various pathologival symptoms) and intensiveness (average number of affected sextants with a certain severity per one patient) of separate pathological symptoms including a general proliferation and a course of disease.

Thus UPI which is indicates both in examining children and grown-ups helps to chatacterize various kinds of the pathological process and represent a clinical picture in dynamics of treatment plus its final results.

Periodontal index (Russel) [PI]

PI score per individual is determined by summing all of the tooth scores and dividing by the number of teeth examined

The periodontal Index (Russel)

Score Criteria and Scoring	Additional X-ray Criteria
0 - Negative	Radiographic appearance is essentially normal
1 - Mild gingivitis. There is an avert area of inflammation in the free gingivae, but this area does not circumscribe the tooth 2 - Gingivitis. Inflammation completely circumscribes the tooth but there is no apparent break in the epithelial attachment	

4 - Used when radiographs are available	There is an early, notchlike resorption of the alveolar crest
6 - Gingivitis with a pocket formation. The epithelial attachment has been broken and there is a pocket. The tooth is firm has not drifted, normal mastication	There is a horizontal bone loss involving the entire alveolar crest, up to half of the length of the tooth root
8 - Advanced destruction with a loss of masticatory function. The tooth may be loose, may have drifted, may sound dull on percussion with a metallic instrument, may be depressible in its socket	There is an advanced bone loss, involving more than one-half of the length of the tooth root, or a definite infrabony pocket with widening of the periodontal ligament. There may be a root resorption, or rare fracture at the apex

Rule: when in doubt, assign the lesser scores

Periodontal Index Score per person = $\frac{\text{summary of individual scores}}{\text{number of teeth present}}$

Clinical Condition	Group PI Scores	Stage of Disease
Clinically normal supportive tissues. Simple gingivitis	0 to 0,2 0,3 to 0,9	Reversible

Beginning of the destructive periodontal disease	0,7 to 1,9	
Established destructive periodontal disease	1,6 to 5,0	Irreversible
Terminal disease	3,8 to 8,0	

3.3. Indices Used to Assess the Amount of Plaque and Calculus

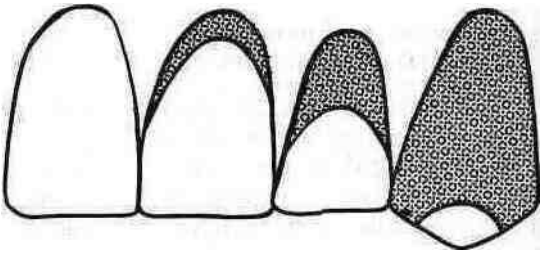
Simplified Oral Hygiene Index (Greene and Vermillion)

In the early development of the indices used to measure gingivitis and periodontal disease, it became apparent that the data lacked meaning or significance unless the level of the oral hygiene or cleanliness was evaluated as a separate component. The lack of a simple, objective set of criteria that minimize the examiner variability prompted Greene and Vermillion to develop the Oral Hygiene Index (OHI). Their goal was to develop a measuring technique that could be used in "studying the epidemiology of periodontal disease and oral calculus, when assessing tooth-brushing efficiency, and when evaluating the dental health practices of a community and the immediate as well as the long-term effects of dental health education" programs. Realizing that it was not necessary or practical to assess all of the teeth to determine a person's level of oral cleanliness, Greene and Vermillion selected six index tooth surfaces that were representatives of all anterior and posterior segments of the mastication (Simplified Oral Hygiene Index).

The OHI-S consists of two components;

a Debris Index (DI-S) and a Calculus Index (CI-S). Each component is assessed on a scale of 0 to 3. Only a mouth mirror and shepherd's crook or sickle-type dental explorer, and no disclosing agent, are used for the examination. The six tooth surfaces examined in the OHI-S are the facial surfaces of the teeth numbered 16, 11, 26, and 31 and the lingual surfaces of the teeth numbered 36 and 46. Each tooth surface is divided horizontally into gingival, middle, and incisal thirds.

For the Debris Index (DI-S)



0 1 2 3

Criteria for Scoring Oral Debris (DI-S) Component of OHI-S.

0 – No debris or stain present.

1 – Soft debris covering not more than one third of the tooth surface, or the presence of extrinsic stains without other debris regardless of surface area covered.

2 – Soft debris covering more than one third but not more than two thirds of the exposed tooth surface.

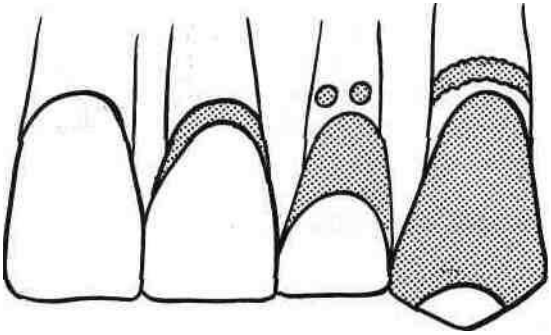
3 – Soft debris covering more than two thirds of the exposed tooth surface.

Explorer is placed on the incisal third of the tooth and moved toward the gingival third. The Debris Index score per person is obtained by totaling the debris score per tooth surface and dividing by the number of surfaces examined.

The Calculus Index (CI-S) is performed by gently placing a dental explorer into the distal gingival crevice and drawing it subgingivally from the distal contact area to the mesial contact area (i.e., one half of a tooth's circumference is considered a scoring unit). The Calculus Index score per person is obtained by totaling the calculus score per tooth surface and dividing by the number of surfaces examined. The OHI-S score per person is the total of the DI-S and CI-S scores per person.

The clinical levels of oral cleanliness for debris that can be associated with group Simplified Debris Index scores are as follows:

Good 0.3 to 0.6
Fair 0.7 to 1.8
Poor 1.9 to 3.0.



1 – Supragingival calculus covering not more than one third of the exposed tooth surface.

2 – Supragingival calculus covering more than one third but not more than two thirds of the exposed tooth surface or the presence of individual flecks of sub-gingival calculus around the cervical portion of the tooth or both.

3 – Supragingival calculus covering more than two thirds of the exposed tooth surface or a continuous heavy band of subgingival calculus around the cervical portion of a tooth or both.

The clinical levels of the oral hygiene that can be associated with group OHI-S scores are as follows; Good 0.3 to 0.6

Fair 0.7 to 1.8

Poor 1.9 to 3.0

The major strength of the OHI-S is in its use in epidemiologic surveys and in evaluating dental health education programs (longitudinal). It can also be used to evaluate an individual's level of the oral cleanliness and, to a more limited extent, can be used in clinical trials. The index is easy to use.

Questions

1. What are the criteria of the estimation of oral; hygiene indeces?
2. How can indices used in studying periodontal problems be divided?
3. Make a description of PMA Index in Parma modification.
4. What is detected by bleeding index (Denega, 1996) and how is it scored?
5. What are the criteria for scoring of gingival index (Silness-Loe)?
6. What indices show the degree of a periodontal destruction. How is the periodontal index Russel calculated?
7. What is known about the CPITN index?
8. Describe the evaluation of a simplifired oral hygieve index (Green and Vermillion).

4. CLINICAL EXAMINATION AND DIAGNOSIS

4.1. Examination of the Oral Tissues

Chief complaints: bleeding gums, loose teeth, spreading of the teeth with the appearance of spaces where none existed before, foul taste in the mouth, itchy feeling in the gums, relieved by a digging with a toothpick, constant dull gnawing pain, dull pain after eating, deep radiating pains in the jaws, acute throbbing pain, sensitivity to percussion, sensitivity to heat and cold, burning sensation in the gums, extreme sensitivity to inhaled air.

Overall appraisal of the patient (Dental history)

Systemic history: Patient's complains of gastrointestinal diseases, liver disease, diabetes, possibility of occupational disease, history of allergy, sensitivity to drugs and dental materials, rheumatic fever, heart disease, hypertension, angina pectoris, myocardial infarction, nephritis, abnormal bleeding tendencies, excessive bruising, infectious diseases.

Oral examination

- oral hygiene
- mouth odors
- saliva
- lips
- oral mucosa
- floor of the mouth
- tongue
- palate
- oropharyngeal region

Examination of the teeth

- hypersensitivity

- proximal contact relations
- tooth mobility
- sensitivity to percussion
- pathologic migration of the teeth
- the dentition with the jaws closed
- examination of functional occlusal relationships
- the temporomandibular joint

Examination of the Periodontium, with a periodontal probing as the main method of clinical examination

- plaque and calculus
- gingiva (change in the consistency, surface texture and position of the gingiva, changes in gingival contour, the presence of „Stilman's Clefts" and McCall Festoons)
- periodontal pockets
- suppuration
- amount of attached gingiva
- sinus formation
- alveolar bone loss
- trauma from occlusion

4.2. Additional Methods in the Diagnosis of Periodontal Diseases

Investigation of the periodontal capillary firmness

Negative pressure (20-40 mm) is applied to the oral mucosa. Normally the time of hematoma formation is 50-60 sec in front region and 70-80 sec in the region of molars. In periodontal inflammation permeability of capillaries increases and their firmness is reduced. The time of hematoma formation is 15-25 sec in gingivitis and 5-10 sec in periodontitis.

Vacuum hematomas have also a therapeutical effect like autohaemotherapy, they stimulate the metabolism and regeneration processes in periodontal tissues.

Biomicroscopy – vital investigation of the condition of a periodontal tissue with a special microscopy.

Reography – method of diagnostic of blood supply in periodontal tissues. It is based on registration of electric resistance of the periodontal tissues while passing through them of an alternating current.

Poliarography of periodontal tissues – the method characterizing oxidation processes in the tissues.

Cytological methods of diagnostics are based on the investigation and analysing the contents of periodontal pockets. They allows to estimate the level of phagocytosis and its completion while indicating quality and quantity of PMN and other cells like lymphocytes, polyblasts, plasmatic cells and epithelial cells.

Exfoliative cytology – method which allows to check the barrier function of oral mucsa. Keratinization index can be calculated as the ratio of keratinized cells to all cells in the field of vision multiplied to 100. Decrease of keratinization indicates to the lowering of the host defence.

Microbiological investigations

Only a small percentage of the more than 500 bacterial species that have been isolated within peridontal pockets are thought to be associated with periodontal disease. Pathogens implicated in periodontal disease belong to facultative anaerobes.

Because of the potential for bacterial resistance as well as the different sensitivities of bacteria to a given antibiotic, antimicrobial sensitivity testing should be considered before commencing antibiotic therapy.

Absorption of microorganisms by epithelial cells of gingiva – a test where estimation is made in 100 cells. Percentage of positive absorption 70% indicates to good functional condition of the body, 31-69% - satisfactory condition, 30% and lower - unsatisfactory condition.

The radiograph is widely used in the diagnosis of periodontal disease. It is highly informative method for the investigation of the alveolar bone structure. Because the facial and lingual bony plates are obscured by the relatively dense root structure, radiographic evaluation of bone changes in periodontal disease is based upon the apperance of the interdental septa.

Because radiograph does not reveal minor destructive changes in bone, periodontal disease that produces even slight radiographic changes has progressed beyond its earliest stages. The earliest signs of periodontal disease must therefore be detected clinically.

Normal position of the apex of interdental septa is situated of the same level as the neck of the tooth (enamel-cementum) junction or 1-2 mm lower.

Lamina dura, seen as dense uninterrupted line. Fuzziness and a break in the continuity of the lamina dura at the mesial or distal aspect of the crest of the interdental septum are the earliest radiographic changes in periodontitis.

In periodontal disease the interdental septa undergo changes that effect the lamina dura, the crestal radiodensity, the size and shape of the medullary spaces, and the height and contour of the bone.

The interdental septa may be reduced in height, with the crest horizontal and perpendicular to the long axis of the adjacent teeth, or they may present angular defects. The former condition is called horizontal bone loss and the latter, angular or vertical bone loss. Dense cortical plates on the facial and lingual surfaces of the interdental septa obscure destruction

which occurs in the intervening cancellous bone. This means that it is possible to have a deep crater in the bone between the facial and lingual plates without radiographic indication of its presence. In order for destruction of the interproximal cancellous bone to be recorded radiographically, the cortical bone must be involved. Reduction of only 0,5 or 1,0 mm in the thickness of the cortical plate is sufficient to permit radiographic visualization of destruction of the inner cancellous trabecular.

Trauma from occlusion can produce radiographically defectable changes in the lamina dura, in the morphology of the alveolar crest, in the width of the periodontal space, and in the density of the surrounding cancellous bone.

Questions

1. What is the sequence of main clinical examination in periodontal patients?
2. What additional methods of diagnostic are used in periodontology?
3. What are the main structures investigated radiographically?

5. ETIOLOGY AND PATHOGENESIS OF THE PERIODONTAL DISEASE

5.1. Pathogens Implicated in Periodontal Disease

Only a small percentage of the bacterial species that have been isolated within peridontal pockets are thought to be associated with periodontal disease. Pathogens implicated in periodontal disease belong to facultative anaerobes and

include: Actinobacillus actinomycetemcomitans, Porhyromonas gingivalis, Treponema denticoli, Bacteroides forsythus, Prevotella intermedia, Enbacterium nodatum, Spirochetes associated with acute necrotizing gingivitis.

The process of tissue destruction results from the elaboration of bacterial substances that directly or indirectly cause degradation of the periodontal tissues. Thus, virulence properties can be broadly categorized into factors that enable a bacterial species to colonize and invade host tissues, and factors that enable a bacterial species to directly or indirectly cause host damage. Bacterial species that have been identified as capable of tissue invasionare strongly associated with diseased sites. The ability to invade has been proposed by Loesche W.J. (Loesche W.J. Bacterial mediators in periodontal disease. Clin.Infect.Dis.16 (suppl 4): S203. 1993) as a key factor distinguishing pathogenic from nonpathogenic gram-negative species. For bacteria to survive in the periodontal environment, they must evade the host mechanisms involved in bacterial clearing.

The production of immunoglobulin-degrading proteases by (P.gingivalis, P.intermedia, P.melaninogenica, Capnocytophaga sp.) may counteract these host defenses. Some bacteria produce substances suppressing the activity of polymorphonuclear leucocytes and lymphocytes. A.actinomycetemcomitans produces leukotoxin inhibiting the function of PMNs and killing of mature B and T cells, P.gingivalis inhibites of superoxide production by PMNs (Socransky S.S., Haffejee A.D., 1991). Pathogenic bacteria also produce enzymes capable of degrading host tissues: P. Gingivalis and A.actinomycetemcomitans produce collagenase, trypsin-like enzyme; T.denticola and P.gingivalis – keratinase; arylsulfatase is produced by C.rectus; neuraminidase – by P.gingivalis, B.forsythus, P.melaninogenica; fibronectin-degrading enzyme is produced

by both – P.gingivalis and P.intermedia; phospholipase A – by P.intermedia and P.melaninogenica (Socransky S.S., Haffejee A.D., 1993). Some bacterial products inhibit the growth or alter the metabolism of host tissue cells, these are: ammonia, volatile sulfur compounds, fatty acids, peptides, indole.

Macrophages, monocytes exposed to bacterial endotoxin (lipopolysaccharide) release interleukin-1, tumor necrosis factor and prostaglandins. These host-derived cytokines stimulate bone resorption, activate or inhibit other host immune cells.

5.2. Influence of Systemic Diseases on the Periodontium

In spite of the well-grounded role of periodontal pathogens in the development of inflammatory-destructive processes, the state of antiinfectious immunity of the host is not less important in periodontitis. The state of immune system determines the possibility of realization of microbiota's pathogenic influence on the periodontal tissues. In this context systemic immunity is especially important, it defines the intensity of local immunity. Because somatic pathology is an often finding in patients with generalized periodontitis. Diseases of gastro-intestinal tract, hepato-biliary system, throat and nose diseases and chronic foci of infection in other organs as well as lingering stress situations lead to the systemic changes of the immune system and that is why these patients have lowered general reactivity of the organism, what allows periodontal pathogens to induce the development of inflammatory-destructive processes in the periodontium.

The condition of the periodontium is strongly influenced by the nutritional diseases, endocrine diseases, modification of sex hormones, hematologic diseases, immunodeficiency disorders, other systemic diseases

(cardiovascular diseases, metal intoxications, stress reactions). Some of the chronic diseases may predispose the patient to periodontal disease by imparing tissue resistance to local irritants and creating a tendency towards gingivitis and alveolar bone loss.

The majority of opinions and research findings on the effects of nutrition on oral and periodontal tissues point to the following (Fermin A Garranza, Clinical Periodontology, 1996):

1. There are nutritional deficiencies that produce changes in the oral cavity. These changes include alterations of the lips, oral mucosa, and bone, as well as of the periodontal tissues. These changes are considered to be periodontal or oral manifestations of nutritional disease.

2. There are no nutritional deficiencies that by themselves can cause gingivitis or periodontal pockets. There are, however, nutritional deficiencies that can affect the condition of the periodontium and thereby aggravate the injurious effects of local irritants and excessive occlusal forces. Theoretically, it can be assumed that there may be a "border zone" in which local irritants of insufficient severity can cause gingival and periodontal disorders if their effect on the periodontium is aggravated by nutritional deficiencies (F.A. Garranza, 1996).

Numerous experiments on animals have shown that the physical character of the diet may play some role in the accumulation of plaque and the development of gingivitis. Soft diets, although nutritionally adequate, may lead to plaque and calculus formation. Hard and fibrous foods provide surface cleansing action and stimulation, which result in less plaque and gingivitis, even if the diet is nutritionally inadequate.

In humans, however, studies have been unable to demonstrate reduced plaque formation when hard foods are consumed. The discrepancy may be related to differences in tooth anatomy and to the fact that hard foods are fed to

experimental animals as the only diet, whereas humans also consume soft foods. Human diets also have a high sucrose content, which favors the production of a thick plaque.

By its effects on the oral bacteria, the diet may influence the relative distribution of types of organisms, their metabolic activity, and their pathogenic potential, which in turn affect the occurrence and severity of oral disease.

Sources of nutrients for the microorganisms can be endogenous and exogenous. Among the exogenous factors, the influence of the sugar content of the diet has been extensively studied, and it has been demonstrated that the amount and type of carbohydrates in the diet and the frequency of intake can influence bacterial growth. Attachment and subsequent colonization of the tooth surface by certain microorganisms may also be made possible by components of the diet (F.A. Garranza, 1996).

Vitamins Deficiencies

Deficiency of vitamin A results in ocular manifestations and keratinizing metaplasia of the epithelium.

The following periodontal changes have been reported in vitamin A-deficient rats: hyperplasia and hyperkeratinization of the gingival epithelium with proliferation of the junctional epithelium, and retardation of gingival wound healing. In the presence of local irritation, vitamin A-deficient rats develop periodontal pockets that are deeper than those in non-vitamin A-deficient animals and exhibit associated epithelial hyperkeratosis.There is little information regarding the effects of vitamin A deficiency on the oral structures in humans. Several epidemiologic studies have failed to demonstrate any relation between this vitamin and periodontal disease (Frandsen A.M., 1963).

Vitamin D, or calciferol, is essential for the absorption of calcium from the gastrointestinal tract and for the maintenance of the calcium-phosphorus balance. Deficiency in

vitamin D and/or imbalance in calcium-phosphorus intake results in rickets in the very young and osteomalacia in adults.

The effect of such deficiency or imbalance on the periodontal tissues of young dogs results in osteoporosis of alveolar bone; osteoid that forms at a normal rate but remains uncalcified; failure of osteoid to resorb, which leads to its excessive accumulation; reduction in the width of the periodontal space; a normal rate of cementum formation, but defective calcification and some cementum resorption; and distortion of the growth pattern of alveolar bone.

In osteomalacic animals, there is a rapid, generalized, severe osteoclastic resorption of alveolar bone, proliferation of fibroblasts that replace bone and marrow, and new bone formation around the remnants of unresorbed bony trabeculae.

Radiographically, there is a generalized partial to complete disappearance of the lamina dura and reduced density of the supporting bone, loss of trabeculae, increased radiolucency of the trabecular interstices, and increased prominence of the remaining trabeculae. Microscopic and radiographic changes in the periodontium are almost identical with those seen in experimentally induced hyperparathyroidism (Becks H., Collins D.A., Feutog R.M., 1946).

No relationship has been demonstrated between deficiencies in vitamin E and oral disease, but systemic vitamin E appears to accelerate gingival wound healing in the rat.

B Complex Deficiency. The vitamin B complex includes thiamine, riboflavin, niacin, pyridoxine (B6), biotin, folic acid, and cobalamin (B12). Oral disease is rarely due to a deficiency in just one component of the B complex group; the deficiency is generally multiple.

Oral changes common to B complex deficiencies are gingivitis, glossitis, glossodynia, angular cheilitis, and inflammation of the entire oral mucosa. The gingivitis in

vitamin B deficiencies is nonspecific, as it is caused mostly by bacterial plaque.

The following oral changes are typical to thiamine deficiency: hypersensitivity of the oral mucosa; minute vesicles (simulating herpes) on the buccal mucosa, under the tongue, or on the palate; and erosion of the oral mucosa.

The symptoms of riboflavin deficiency (ariboflavinosis) include glossitis, angular cheilitis, seborrheic dermatitis, and a superficial vascularizing keratitis. The glossitis is characterized by a magenta discoloration and atrophy of the papillae. In mild to moderate cases, the dorsum exhibits a patchy atrophy of the lingual papillae. In severe deficiency, the entire dorsum is flat, with a dry and often fissured surface.

Angular cheilitis begins as an inflammation of the commissure of the lips, followed by erosion, ulceration, and fissuring. Riboflavin deficiency is not the only cause of angular cheilitis. Loss of vertical dimension, together with drooling of saliva into the angles of the lips, may produce a condition similar to angular cheilitis. Candidiasis may develop in the commissures of debilitated persons.

Changes observed in riboflavin-deficient animals include severe lesions of the gingivae, periodontal tissues, and oral mucosa (including noma).

Niacin deficiency results in pellagra, which is characterized by dermatitis, gastrointestinal disturbances, neurologic and mental disturbances (dermatitis, diarrhea, and dementia), glossitis, gingivitis, and generalized stomatitis.

Glossitis and stomatitis may be the earliest clinical signs of niacin deficiency. The gingiva may be involved in aniacinosis with or without tongue changes. The most frequent finding is acute necrotizing ulcerative gingivitis, usually in areas of local irritation.

Oral manifestations of vitamin B complex and niacin deficiency in experimental animals include black tongue and

gingival inflammation with destruction of the gingiva, periodontal ligament, and alveolar bone. Necrosis of the gingiva and other oral tissues and leukopenia are terminal features of niacin deficiency in experimental animals.

Folic acid deficiency results in macrocytic anaemia with megaloblastic erythropoiesis, with oral changes and gastrointestinal lesions, diarrhea, and intestinal malabsorption (Afonsky D., 1995)

Folic acid-deficient animals demonstrate necrosis of the gingiva, periodontal ligament, and alveolar bone without inflammation. The absence of inflammation is the result of deficiency-induced granulocytopenia. In humans with sprue and other folic acid deficiency states, there is a generalized stomatitis, which may be accompanied by ulcerated glossitis and cheilitis. Ulcerative stomatitis is an early indication of the toxic effect of folic acid antagonists used in the treatment of leukemia (Shaw J.H., 1962).

In a series of human studies, a significant reduction of gingival inflammation has been reported after systemic or local use of folic acid, when compared with placebo. This reduction occurred with no change in plaque accumulation. The same authors have postulated that the gingival changes associated with pregnancy and oral contraceptives may be partly related to suboptimal levels of folic acid in the gingiva. In a clinical study of pregnant women, a reduction in gingival inflammation occurred with the use of topical folate mouth rinses; no change was found with systemic folic acid. A relationship has also been assumed between phenytoin-induced gingival overgrowth and folic acid, based on the interference of folic acid absorption and utilization by phenytoin (Vogel R., 1980).

Severe vitamin C deficiency in humans results in scurvy, a disease characterized by hemorrhagic diathesis and retardation of wound healing. Vitamin C is required in the human diet but not in that of other animals except other

primates, guinea pigs, and some rare flying mammals. Vitamin C is abundant in fruits and vegetables. Scurvy is uncommon in countries that have adequate food supplies, but it may appear in infants in their first year of life if formulas are not fortified with vitamins and in the very elderly, especially those living alone and on restricted diets. Alcoholism also may predispose an individual to scurvy.

Clinical manifestations of scurvy include hemorrhagic lesions into the muscles of the extremities, the joints, and sometimes the nail beds; petechial hemorrhages, often around hair follicles; increased susceptibility to infections; and impaired wound healing. Bleeding, swollen gingivae, and loosened teeth are also common features of scurvy.

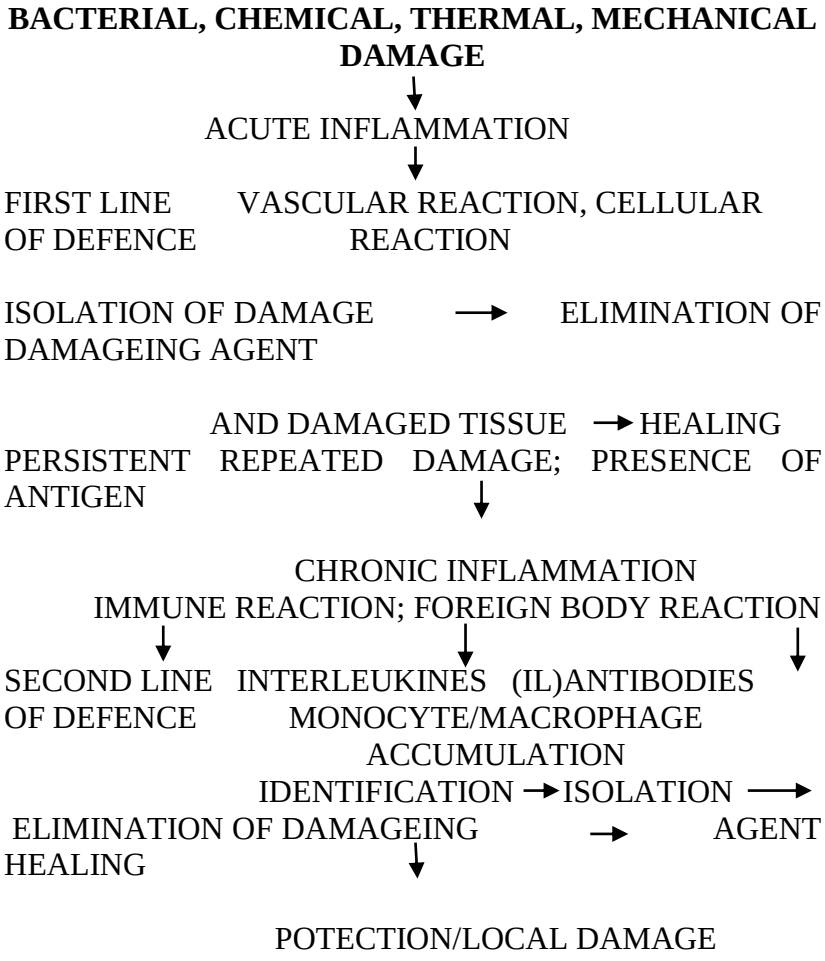
Vitamin C deficiency (scurvy) results in defective formation and maintenance of collagen, retardation or cessation of osteoid formation, and impaired osteoblastic function. Vitamin C deficiency is also characterized by increased capillary permeability, susceptibility to traumatic hemorrhages, hyporeactivity of the contractile elements of the peripheral blood vessels, and sluggishness of blood flow (Cotran R.S., Kumar V. et al., 1989).

Possible Etiologic Relationships Between Ascorbic Acid and Periodontal Disease. It has been suggested that ascorbic acid may play a role in periodontal disease influencing the metabolism of collagen within the periodontium, thereby affecting the ability of the tissue to regenerate and repair itself. Ascorbic acid deficiency interferes with bone formation. Changes in alveolar bone and other bones are the result of failure of the osteoblasts to form osteoid. These changes take place very late in the deficiency state. Osteoporosis of alveolar bone can be a result of increased osteoclastic resorption and is not associated with periodontal pocket formation. Optimal levels of vitamin C, therefore, would maintain the epithelium's barrier function to bacterial products. Increasing levels of

ascorbic acid enhance both the chemo-tactic and the migratory action of leukocytes without influencing their phagocytic activity. Megadoses of vitamin C seem to impair the bactericidal activity of leukocytes. Depletion of vitamin C may interfere with the ecologic equilibrium of bacteria in plaque and thus increase its patho genicity. However, there is no evidence that demonstrates this effect (F.A. Garranza, 1996).

The exaggerated destruction results partly from inability to marshal a defensive delimiting connective tissue barrier reaction to the inflammation and partly from destructive tendencies caused by the deficiency itself, including inhibition of fibroblast formation and differentiation to osteoblasts and impaired formation of collagen and mucopolysaccharide ground substance.

5.3. Pathogenesis of Plaque Associated Periodontal Disease



Schaematic outline depicting the acute inflammatory process as the first, and the chronic inflammatory process as the second line of host defence.

Adapted from Lindhe J. Textbook of Clinical Periodontology.

Note that in both conditions there is a delicate balance between protection and local tissue damage. This means that protection of the organism is achieved at the price of local damage.

Basic phenomena in inflammation

Inflammatory lesions occurring in the gingiva are no different from similar lesions in other tissue compartments. The location, extension and composition of gingival inflammatory lesions are influenced, however, by the morphology and physiology of the tissues of the dentogingival region.

The acute inflammatory reaction may be regarded as the first line of defence in the tissue following irritation or damage, while the so-called „chronic" inflammatory reaction may be looked upon as the second line of defence

Cytotoxic reaction

The microbiota of the tooth surface contains and releases enzymes such as proteases, hyaluronidases, collagenases, etc. Such enzymes could be the direct cause of epithelial and connective tissue damage, and be, at least partly, responsible for tissue destruction in gingival lesions.

A lipopolysaccharide, endotoxin from Gram negative bacteria, may activate the complement system (induce acute inflammation) and is, in addition, cytotoxic to host cells. Endotoxin could, therefore, contribute some of the tissue changes observed in gingival lesions. It has been suggested that teichoic acid from Gram positive bacteria has a similar effect.

It was recently demonstrated that a leukotoxin produced by a Gram negative rod, Actinobacillus actinomycetemcomitans, has the capacity to interfere with the vitality and function of phagocytosing leukocytes.

Immune reaction

Most of the substances produced by plaque microbiota are antigenic, therefore elicit a general and a local immune reaction. Immune reactions protect gingival tissue against antigens produced by plaque microbiota.

Antibody mediated reaction

The inflammatory cell infiltrate of established and advanced gingival lesions is characterized by the presence of large numbers of plasma cells. Most plasma cells produce immunoglobulins of the IgG class. A small number of plasma cells produces IgA and some also IgM. Antibodies may participate in the defence of gingival tissue by neutralizing toxins and enzymes from plaque. Furthermore, antibodies may decrease penetration of tissue by bacterial products (antigens) from the gingival sulcus/-pocket.

Antibodies, both within the plasma cells and extracellularly, are present in the connective tissue and the extracellular space of the junctional epithelium. When an antigen and an antibody combine to protein aggregates (immune complexes) in the tissue, complement is activated and factors chemotactic to neutrophilic granulocytes are formed. In particular, activation of the fifth component of complement, C5, releases such chemotactic factors. Activation of complement also releases factors which cause increased vascular permeability. Immune complexes deposited in gingival tissue may thereby cause increased migration and accumulation of neutrophilic granulocytes and increased exudation of plasma proteins from the adjacent vasculature. Neutrophils have the capacity to phagocytose the antigen/antibody complex. In the process of phagocytosis, however, neutrophilic granulocytes will release some of their lysosomal enzymes into the surrounding tissue. These lysosomal enzymes can in turn cause tissue damage and increase the intensity of inflammatory reaction. Thus, in an

attempt to defend the tissue against antigenic material a reaction is elicited which may cause tissue damage.

Activation of the complement system can also stimulate neutrophilic granulocytes (and macrophages) to produce and release prostaglandins. Prostaglandins are proteins which, not only can enhance vascular permeability but also activate osteoclasts. The immune system, therefore, through complement activation can (1) mediate vascular and cellular reactions and (2) cause or induce bone resorption. The complement system may also be activated by enzymes and endotoxin from subgingival microbiota. Complement activated in this manner has a similar effect on neutrophils as described above. The relative importance of these mechanisms in gingival inflammation is presently not understood. However, it is likely that they all participate in gingivitis and contribute to tissue destruction.

Cell mediated immune reaction

The presence of lymphocytes in gingival lesions indicates that cell mediated immune reactions can occur in the periodontium.

As opposed to normal gingiva, gingivitis and advanced periodontal disease induce circulating lymphocytes with an increased ability to react to antigens from certain plaque forming microorganisms.

In such reactions lymphocytes, upon exposure to antigens to which they are sensitized, produce interleukines (IL). Such interleukines (IL) can cause migration of inflammatory cells from the vascular system and induce increased vascular permeability. Other, interleukines (IL) are cytotoxic and can cause damage to connective tissue cells. Thus, if the cell mediated immune reaction takes place close to fibroblasts, these cells may degenerate. Degenerated fibroblasts have a decreased capacity to produce collagen and matrix substance. Of particular significance is the ability of some

interleukines (IL) to mediate accumulation of monocytes/macrophages and stimulate these phagocytosing cells to an increased synthetic activity. By this stimulation the capacity of macrophages to produce and release lysosomal enzymes and prostaglandins into surrounding tissue is enhanced. The resultant tissue destruction may be similar to that produced by the release of lysosomal material from neutrophilic granulocytes. Some Interleukines (IL) released from sensitized lymphocytes, activate osteoclasts and initiate bone resorption.

The host response to exogenous antigen involves a complex series of interactions amongst macrophages, lymphocytes and other cells of inflammatory system. Many, if not all, of these interactions are mediated by small molecular weight polypeptides known as cytokines, although the earlier term lymphokine remains in common usage amongst immunologists for those factors produced by lymphocytes. These key mediators of the host response include the interleukines, tumor necrosis factors and interferons. Interleukin-1, -2, -4 and -6 are considered to play an important role in the pathogenesis of periodontal diseases (Meikle et al., 1989).

Originally mononuclear phagocytes were thought to be a unique source of interleukin-1 (IL-1) but a wide variety of cell types including keratinocytes (Luger et al., 1981), fibroblasts (Health et al., 1985), endothelial cells (Miossec et al., 1986) and osteoblasts (Hanazawa et al., 1987) can produce IL-1 like molecules.

IL-1 has been shown to enhance various immune responses in vitro, including Blymphocyte differentiation, antibody secretion and T lymphocyte proliferation through the induction of IL-2 production. IL-1 can also stimulate non-leucocytic cell populations, mediating several non-immunological events both in vivo and in vitro; these include

acute phase protein synthesis, endogenous pyrogen induced fever, fibroblast proliferation, bone resorption and collagenase and prostaglandin E2 production by fibroblasts and chondrocytes (Dinarello, 1985; Meikle et al.,1986).

IL-1 is functionally related to the tumor necrosis factors. The tumor necrosis factors TNF-α and TNF-ß are products of activated macrophages and lymphocytes respectively. Although originally defined by their cytotoxic action, it has become apparent with the widespread availability of recombinant TNF-α that the TNFs are potent immunoregulatory molecules sharing multiple overlapping biological activities with IL-1 (Le & Vilcek, 1987). IL-1ß and TNF-α can be detected in monocyte culture supernatants from periodontitis patients, and lipopolysaccharide (LPS)-stimulated monocytes from periodontitis patients release significantly more IL-1ß □and TNF-α □than control subjects (Mc Farlane et al., 1989).

IL-2 is the protein produced by activated T lymphocytes. Recent evidence suggests that both human CD4+(helper) and CD8+(supressor) T-cell clones can produce IL-2 (Paliard et al., 1988). In common with all cytokines IL-2 exerts pleiotrophic effects via specific receptors present in thymocytes, activated lymphocytes, NK cells, monocytes and endothelial cells (Kroemer & Wiek, 1989). IL-2 plays an essential role in T-cell, B-cell, NK effector cell function and thymocyte differentiation and the two most effective immunosupressive agents known, cyclosporin and the glycocorticoids exert their effect through a selective inhibition of IL-2 production.

Recent investigations of T-cell function in periodontal diseases have focused on the spontaneous proliferative response (SPR) of peripheral blood lymphocytes stimulated in vitro by autologous non-T-cells in the absence of added antigen. The SPR has been shown to be depressed in patients

with periodontitis relative to healthy control subjects, but returns to normal following treatment (Tew et al., 1983; Suzuki et al., 1984; Evans et al., 1989). Deficient in vitro cell-mediated responses have also been reported in several diseases including systemic lupus erythematosis (SLE), Sjogren"s syndrome and rheumatoid arthritis (RA), and have been attributed to an incapacity of helper T-cells to produce IL-2 (Kitas et al., 1989).

IL-4 has now been shown to be secreted by human B-cells as well as fetal and adult immature mouse thymocytes (Ransom et al., 1987; Zlotnik et al., 1987). IL-4 has been found to possess a wide spectrum of biological activities in vitro on cells of hemopoietic lineage. During the pathogenesis of periodontal diseases, as in other inflammatory diseases, there occurs a considerable infiltration of both T-cells and B-cells in to the effected tissue (Seymour & Greenspan, 1979; Johannessen et al., 1986). These cells produce an array of inflammatory mediators, including IL-4. This cytokine would then be an important factor in the regulation of both early B-cell differentiation and proliferation as well as being involved in Ig class switching and enhancing the formation of T/B cell and T cell/macrophage conjugates through an increase in class II MHC (major histocompatibility complex) antigen expression. This lymphokine may also have a possible physiologic role in maintaining a pool of immature T cells by promoting self renewal of a subset of stem cells within the thymus.

The activities of IL-4 in vitro have been shown to be regulated by interferon γ (IFN-γ). This product of activated lymphocytes inhibits the IL-4 mediated proliferation of immature Lyt-/L3T4-lymphocytes (Ransom et al 1987), the IL-4 stimulated switching to IgG1 and IgE (Finkelman et.al., 1988) and the IL-4 mediated proliferation of resting B-cells (Rabin et al., 1986). IFN-γ is known to be a potent inducer of

class I and II MHC antigen expression on macrophages and the IL-4 stimulation of class II MHC antigen expression by human monocytes has been found to be IFN-γ dependent whereas stimulation of class III antigens was not (Littman et al., 1989). The expression of IFN-γ and IL-4 by murine T-cells has been shown to be mutually exclusive for CD4+subsets (Th1 and Th2 respectively; Mosmann et al., 1986) and this leads to the possibility that selective expression of either of the two subsets could result in a pro-IFN-γ or pro-IL-4 response.

Interleukin-6 (IL-6) was originally characterized by Weissenbach et al. In 1980 as a T-cell derived molecule exhibiting ant-viral activity and it was termed interferon ß$_2$ (IFN-ß$_2$). IL-6 is produced by a variety of cells such as macrophages, fibroblasts, T-cells, hepatocytes, glial cells, vascular endothelial cells (Lots et al., 1988) and B-cells (Horii et al., 1988) amongst others. Its biological activities are many and various and its target cells both lymphoid and non-lymphoid. IL-6 induces the differentiation of activated human B-cells into antibody secreting cells, stimulates the differentiation of human and murine cytolytic T-cells in the presence of IL-2, induces the expression of acute phase proteins by hepatocytes, acts as a co-stimulator of human and murine thymocyte proliferation (Wong & Clark, 1988), stimulates human fibroblast proliferation (Kohase et.al., 1986) and in the mouse it acts as a hemopoietic colony stimulating factor (CSF); Wong 1988).

IL-1, TNF-α and platelet derived growth factor (PDGF) all stimulate an increase in IL-6 production by human fibroblasts (Walther et al., 1988) and more recently IL-1 has been shown to synergise with IL-6 from stimulated human fibroblasts in the thymocyte proliferation assay (Elias et al., 1989). IL-1 is also known to induce the production of IL-6 by murine thymocytes (Helle et al., 1988) and it would appear that

IL-6 is therefore capable of mediating or amplifying some of the tissue effects of IL-1.

The induction of cytolytic T-cells has been proposed to require coordinated cytokine stimulation with IL-2, IFN-γ and IL-6 (Takai et al., 1988) and B cell activation is thought to occur in at least three distinct stages (Muraguchi et al., 1988). Stage one involves the activation of resting cells by IL-4, stage two the promotion of growth and maturation of the activated cells by IL-5 and in stage three, IL-6 induces the terminal differentiation of the B-cells into antibody secreting cells.

Periodontal pathogens mediate connective tissue destruction in periodontal diseases through the ability of antigens from their cell walls to stimulate IL-1 and TNF-α production by circulating mononuclear cells (Meikle 1989). These in turn induce the synthesis of metalloproteinases (MPs, a family of enzymes comprising collagenase, gelatinase (type IV collagenase) and stromelysin, which have the ability to degrade all connective tissue macromolecules at physiologic pH) by resident gingival cells thereby initiating matrix degradation. The progression and extent of tissue degradation is likely to be determined in major part by relative concentrations and half-life of IL-1, TNF-α, and related cytokines, competing molecules such as the IL-1 receptor antagonist, and the suppressive molecules such as transforming growth factor-ß (TGF-ß) and prostaglandin E2 (PGE2). These molecules control levels of latent and active metalloproteinase and urokinase-type plasminogen activator (u-PA) and the availability and concentration of tissue inhibitor of metalloproteinase (TIMP) determines the extent and duration of degradative activity (Page, 1991).

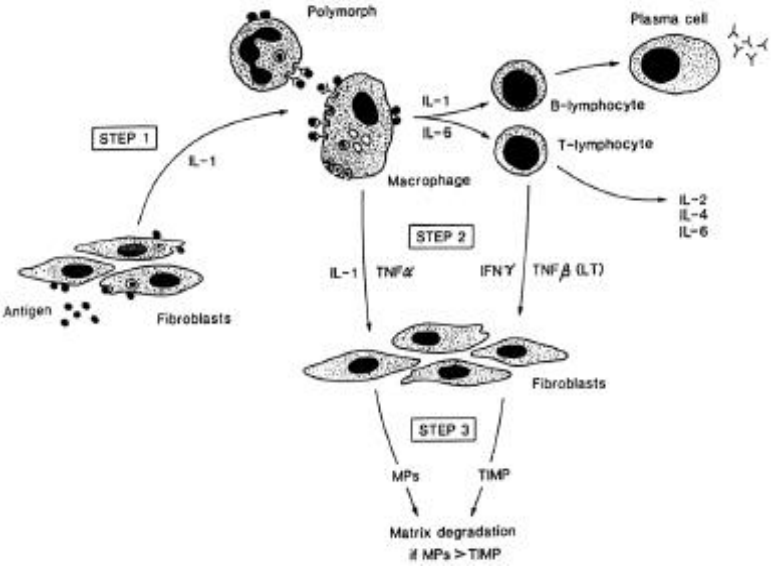
The kallikrein-kinin system and the coagulation cascade are also activated in inflammation (Lerner, 1994). Peptides produced in the kallikrein-kinin system (bradykinin, kallidin) and thrombin, the end product in the coagulation cascade, can

stimulate bone resorption in vitro. Both kinins and thrombin stimulate prostaglandin biosynthesis in bone parallel with the bone resorptive effect. The stimulatory effect of bradykinin on bone resorption is completely lost when prostaglandin response is abolished, whereas thrombin can stimulate bone resorption both via prostaglandin-dependent and independent mechanisms. In addition, bradykinin and thrombin act in concern with IL-1 to synergetically stimulate bone resorption and prostaglandin biosynthesis.

Phospholipase A_2 (PLA_2) is a lipolytic enzyme which hydrolyses cell membrane phospholipids. Considerable increase of PLA_2 activity in periodontal tissues, blood serum and saliva for patients and animals with experimental periodontitis caused by alimentary factor has been revealed (V. Zubachyk, 1997).

PLA_2 was found to be one of the important etiopathogenic factors resulted in periodontitis in experimental animals. This concept has been approved by visual observations of periodontal tissues as well as by biochemical characteristics of inflammation level. The use of PLA_2 in the form of application on gums seems to be a good experimental model for periodontitis.

It has also been found (Lerner, 1994) that one of the acute-phase reactants haptoglobin can stimulate bone resorption in vitro, indicating the possibility of generalized bone loss in chronic inflammatory diseases. Moreover, haptoglobin synergetically potentiates bradykinin-induced and thrombin-induced prostanoid biosynthesis in osteoclasts. These observations indicate that the rate of bone resorption in inflammation -induced bone loss may not be due to a single factor but to the concerted action of several local or systemic factors.



Immunoregulatory cytokins as mediators of connective tissue degradation in periodontal diseases: hypothetical model (Meikle et al., 1989).

IL: Interleukine.

LT: Lymphotoxin (TNF-ß).

IHF: Interferon-y.

MPs: Metalloproteinases.

TIMP: Tissue Inhibitor of metalloproteinases.

FGF: Fibroblast growth factor.

TNF: Tumor necrosis factor.

TGF-R: Transforming growth factor-13.

PDGF: Platelet-derived growth factor.

The cellulal interactions are complex. However, we propose the following sequence of events. In step 1, IL-1 and related cytokines produced by antigen activated gingival fibroblasts are chemotatic for leucocytes. Gingival fibroblasts might also act as tissue localized antigen pressing cells. In step

2, the production of cytokines by antigen activated macrophages stimulates the clonal expantion of T and B lymphocytes: activated T lymphocytes also produced IL-2, IL-4 and IL-6 latent MP production by gingival fibroblasts and their subsequent activation by enzymes such as plasmin/ In step 3, whether matrix degradation occurs or not is determinated by the relative amount of locally produced MPs and their specific inhibitor TIMP (possibly modulated by FGF, PDGF and TGF-R). Gingival fibroblast IL-1 might also stimulate MP production by both autocrine and paracrine mechanisms.

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Questions

1. How oral pathogens influence upon the gingival tissues?
2. What antibody mediated reactions are elicited by antigenic plaque microbiota?
3. What cells take part in cell mediated immune reactions in gingivitis?

6. GINGIVITIS

Gingivitis is an inflammatory process affecting the soft tissues surrounding the teeth. The inflammatory process does not extend into the alveolar bone, periodontal ligament, or cementum. The primary etiologic agent of gingivitis is bacterial plaque.

The role of inflammation in individual cases of gingivitis varies as follows:

1. Inflammation may be the primary and only pathologic change. This is by far the most prevalent type of gingival disease.

2. Inflammation may be a secondary feature, superimposed upon systemically caused gingival disease. For example, inflammation commonly complicates gingival hyperplasia caused by the systemic administration of phenytoin. Gingivitis in patients with gastrointestinal diseases, etc.

3. Inflammation may be the precipitating factor responsible for clinical changes in patients with systemic conditions that of themselves do not produce clinically detectable gingival disease. Gingivitis in pregnancy is an example.

Plaque-associated gingivitis is the most common form of gingivitis and, probably, the most common form of all periodontal diseases. Plaque-associated gingivitis has clinical features including some or all of the following: inflammation, oedema, bleeding upon probing or spontaneous, gingival sensitivity, and itching. However, by definition, no loss of attachment or radiographic loss of bone is associated with gingivitis.

Other factors may modify the course and clinical presentation of gingivitis. These factors permit a classification of gingivitis based upon secondary etiologic factors. The primary agent for most, forms of gingivitis is bacterial plaque.

6.1. Catarral (Simple) Gingivitis

According to distribution of the pathological process it can be localized (confined to the gingiva in relation to a single tooth or group of teeth) and generalized (involving the entire mouth).

Pathology of catarrhal gingivitis

Vascular changes have been described as the first response to initial gingival inflammation – dilatation of capillaries and increased blood flow (clinically, the initial response of the gingiva to bacterial plaque is not apparent). Than proliferation of capillaries and increased formation of capillary loops takes place (clinical signs are erythaema, bleeding upon probing). In chronic gingivitis the blood vessels become engorged, venous return is impaired, and the blood flow becomes sluggish.

The result is a localized gingival anoxemia, which clinically appear in bluish hue of the gums.

Inflammed gingiva histologically is characterized by leukocyte infiltration in connective tissue beneath the junctional epithelium, consisting mainly of lymphocytes but also neutrophils, as well as macrophages, plasma cells and mast cells.

The junctional epithelium becomes densely infiltrated with neutrophils. There is an increase in the amount of collagen destruction.

Key feafure of chronically inflamed gingiva is the increase in number of plasma cells.

The junctional epithelium reveals widened intercellular spaces filled with granular cellular debris, lysosomes derived from disrupted neutrophils, lymphocytes and monocytes. The lysosomes contain acid hydrolases that can destroy tissue components. The junctional epithelium develops riges, protruding info the connective tissue and the basal lamina is destroyed in some areas. There appears to be an inverse relationship between the number of intact collagen bundles and the number of inflammatory cells. Collagenolytic activity is increased in inflamed gingival tissues.

Acute simple gingivitis is characterized by the red colour, loss of surface stippling, puffiness and softening of the

gums, easy bleeding upon probing or spontaneons bleeding and the pain during mastication and palpation.

Chronic simple gingivitis is painless. Gingival bleeding provoked by mechanical trauma (tooth-brushing, food impaction by biting solid foods such as apples) and bleeding upon probing is of great value for the early diagnosis of chronic gingivitis. Gingival bleeding varies in severity, duration and the ease with which it is provoked. The severity of the bleeding and the case with which it is provoked depend upon the intensity of the inflammation (see Bleeding Index).

Chronic gingivitis is a conflict between destructive and reparative changes, with the consistency of the gingiva determined by the relative balance between the two. When inflammatory exudate and tissue degeneration are the predominant microscopic changes - the gingiva is soft, friable and bleeds easily. When fibrosis predominates in the inflammatory process gingiva is firm and nodular.

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

6.2. Acute Necrotizing Ulcerative Gingivitis (ANUG)

The term ***acute necrotizing ulcerative gingivitis*** (ANUG) denotes an inflammatory destructive disease of the gingiva which presents characteristic signs and symptoms. Other terms by which this condition is known are Vincent's infection, acute ulceromembranous gingivitis, trench mouth, trench gums, phagedenic gingivitis, acute ulcerous gingivitis,

acute ulcerative gingivitis, ulcerative gingivitis, ulcerative stomatitis, Vincent's stomatitis, Plaut-Vincent's stomatitis, stomatitis ulcerosa, stomatitis ulcero-membranacea, fusospirillary gingivitis, fusospirillary marginal gingivitis.

Acute necrotizing ulcerative gingivitis (ANUG, Vincent's infection, trench mouth) has several possible secondary etiologic factors. Stress and anxiety are probably significant contributing factors, leading to the lowered resistance of the body. ANUG occurs in young persons, in the case of pre-existing simple gingivitis and poor oral hygiene.

Clinical features

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

Acute necrotizing ulcerative gingivitis is characterized by sudden onset, frequently following an episode of debilitating disease or acute respiratory infection. Occasionally, patients report that it appeared shortly after they had their teeth cleaned. Change in living habits, protracted work without adequate rest, and psychological stress are frequent features of the patient's history.

Characteristic lesions are punched-out, crater-like depressions at the crest of the gingiva that involve the interdental papillae, the marginal gingiva, or both (fig.14). The surface of the gingival craters is covered by a gray, pseudomembranous slough demarcated from the remainder of the gingival mucosa by a pronounced linear erythaema. In some instances, the lesions are denuded of the surface

pseudomembrane, exposing the gingival margin, which is red, shiny, and hemorrhagic. The characteristic lesions progressively destroy the gingiva and underlying periodontal tissues.

A fetid odor, increased salivation, and spontaneous gingival hemorrhage or pronounced bleeding upon the slightest stimulation are additional characteristic clinical signs.

The lesions are extremely sensitive to touch, and the patient complains of a constant radiating, gnawing pain that is intensified by spicy or hot foods and mastication. There is a metallic foul taste and the patient is conscious of an excessive amount of „pasty" saliva. The teeth are characteristically described as feeling like "wooden pegs."

Acute necrotizing ulcerative gingivitis occurs in otherwise disease-free mouths or superimposed upon chronic gingivitis or periodontal pockets. Involvement may be limited to a single tooth or group of teeth, or be widespread throughout the mouth. It is rare in edentulous mouths, but isolated spherical lesions occasionally occur on the soft palate.

Patients are usually ambulatory, with a minimum of systemic complications. Local lymphadenopathy and slight elevation in temperature are common features of the mild and moderate stages of the disease. In severe cases there are marked systemic complications such as high fever, increased pulse rate, leukocytosis, loss of appetite, and general lassitude. Systemic reactions are more severe in children. Insomnia, constipation, gastrointestinal disorders, headache, and mental depression sometimes accompany the condition.

In very rare cases severe sequelae such as the following may occur: noma or gangrenous stomatitis, fusospirochetal meningitis and peritonitis, pulmonary infections, toxemia, and fatal brain abscess.

The clinical course is indefinite. If untreated, it may result in progressive destruction of the periodontium and

denudation of the roots, accompanied by an increase in the severity of toxic systemic complications. It often undergoes a diminution in severity leading to a subacute stage with varying degrees of clinical symptomatology. The disease may subside spontaneously without treatment. Such patients generally present a history of repeated remissions and exacerbations. Recurrence of the condition in previously treated patients is also frequent.

Microscopically, the lesion appears as a nonspecific acute, necrotizing inflammation at the gingival margin involving both the stratified squamous epithelium and the underlying connective tissue. The surface epithelium is destroyed and replaced by a pseudomembranous meshwork of fibrin, necrotic epithelial cells, polymorphonuclear leukocytes, and various types of microorganisms. This is the zone that appears clinically as the surface pseudomembrane. The underlying connective tissue is markedly hyperemic with numerous engorged capillaries and a dense infiltration of polymorphonuclear leukocytes. This acutely inflamed hyperemic zone appears clinically as the linear erythaema beneath the surface pseudomembrane.

The relation of bacteria to the characteristic lesion has been studied with the light microscope and electron microscope. With the former it appears that the exudate on the surface of the necrotic lesion contains microorganisms which morphologically resemble cocci, fusiform bacilli, and spirochetes. The layer between necrotic and living tissue contains enormous numbers of fusiform bacilli and spirochetes in addition to leukocytes and fibrin. Spirochetes invade the underlying living tissue; other organisms seen on the surface are not found there. Some investigators feel that the spirochetes are pushed into the tissue when gingival specimens are removed for microscopic study.

6.3. Hypertrophic Gingivitis

Among the main local etiologic factors – prolonged local irritation (abnormal relationships of adjacent teeth, cervical cavities, overhanging margins of dental restorations, irritations from dental crowns, orthodontic appliances, mouth breathing, nasal obstructions, etc.)

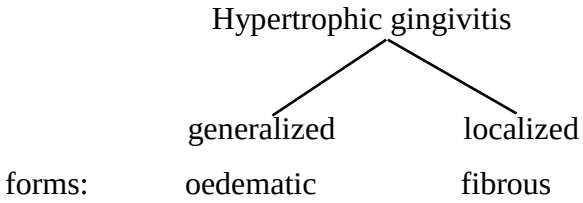
Factors other than local irritation are steroid hormone-influenced gingivitis results from the presence of steroid hormones, which may amplify clinical inflammatory changes of gingivitis. Increased levels of estrogens and progesterones during pregnancy, during adolescence, or in patients who are taking birth control medication may enhance marginal gingival inflammation. Subgingival bacterial growth such as Bacteroides may be enhanced during pregnancy or by steroid therapy. Steroid hormone therapy may result in enhanced gingival tissue response similar to the overall systemic response seen in reproductive organs and mammary glands.

Medication-influenced gingival overgrowth (for example, dilantin hyperplasia or gingival hyperplasia) frequently results in pseudopockets (that is, junctional epithelium at the level of the cementoenamel junction with no loss of attachment). Medications having this potential include phenytoin (dilantin) used for the control of epilepsy, and cyclosporins used for immunosuppressive therapy of renal transplant patients. Fibroblasts in the connective tissue of the periodontium respond with abnormal rates of mitosis in the presence of these (and other) medications resulting in apparently normal cells and fiber and matrix composition. Bacterial plaque is a significant etiologic factor in medication-influenced hyperplastic gingivitis. The pathogenesis of this form of gingivitis is modified by the presence of selected medications (for example, phenytoin, cyclosporin) that results in hyperplastic growth of the marginal gingiva.

Gingival enlargement can be associated with vitamin C deficiency and leukemic enlargement

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

Hypertrophic (Hyperplastic) gingivitis can be classified as follows:



3 stages: light – gums cover 1/3 of the tooth crown, moderate – gums cover not more than 1/2 of the tooth crown, heavy – gums cover 2/3 of the tooth crown or more.

In the case of ***oedematic form*** of hypertrophic gingivitis patients' complaints are: pain and itching in the gums, mouth odour, bleeding when chewing and teeth brushing.

Hypertrophic gingivitis is proceeding as chronic not acute process.

Clinically the gums are bluish red, friable, with intensive bleeding upon probing. Interdental papilla and marginal gingiva are increased in size and produce false periodontal pockets because the dentogingival junction is intact. Intensive dental deposits are present

Fibrous form of hypertrophic gingivitis is characterized by the dense and enlarged gingival tissues with false periodontal pockets and without bleeding. Fibrous form of

gingivitis is painless, the only patient's complaint is the overgrowing of the gums, but the configuration of gum's edge is retained. Fibrous form of gingivitis has to be differentiated from a gingival fibromatosis. In gingival fibromatosis the form of the gums is changed significantly. Gingival surface is uneven and looks like entire mass of tissues surrounding oral and vestibular surfaces of teeth. In the mass of tissues around the teeth in fibromatosis gingival margin and papillae can not be distinguished.

Dental deposits are allways present in the hypertrophic gingivitis.

6.4. Desquamative and Atrophic Gingivitis

Desquamative gingivitis – is the form of gingivitis followed with the increase desquamation of epithelium. Clinically is characterized by the intensive red colour and burning pain in the gums. Desquamative gingivitis is the oral manifestation of some general diseases – gastrointestinal, dermatologic and other systemic diseases, viral infection.

Increase blood levels of estrogens can provoke gingival desquamation. Desquamation gingivitis is revealed in diabetes patients. Among the local etiologic factors of this form of gingivitis – chemical and physical trauma.

The treatment of the main disease is essential in the case of desquamative gingivitis.

Atrophic gingivitis is the term sometimes used to describe gingival recession. Gingival recession is caused locally by some traumatic factors from occlusion, low or high position of the frenulum of the lip upper or lower. Atrophic gingivitis can follow untreated necrotic ulcerative gingivitis. Generalized atrophic gingivitis is one of the signs of dystrophic processes in periodontal tissues.

7. TREATMENT OF GINGIVITIS

7.1. Treatment of Simple Gingivitis

Treatment of simple gingivitis is local and sometimes general, mostly in the case when gingivitis is the sign of some internal diseases.

Local treatment of simple gingivitis is started by the professional hygiene using ultrasound and hand scalers. Mechanical plaque and calculus cleaning and tooth surface polishing should be combined with the use of antiseptic medications.

Groups of antiseptics

Oxidizers:

Solution of Hydrogenii peroxide 0,25%.

Solution of Kalii Permanganatis 1%.

Surface active substances:

solution of Chlorhexidinum bigluconat is 0,2%.

Aethonium solution (0,5%).

Jodine compounds:

Jodinolum – iodine compound with polyvinil alcohol.

Derivaties of nitrofuran – 0,02% Sol. of Furacilinum (1:5000), Sol. of

Furaginum (1:13000).

Herbal medications with antiseptic and antiinflammatory effect are greatly recommended in the treatment of simple gingivitis. Among them:

Alcoholic Solution of Novoimaninum (1%). Has to be dissolved before use in distilled water. Novoimaninum is produced from the stems and flowers of the Hypericem perforatum. Is active against gram positive microorganisms especially staphylococci.

1% linimentum of Sanguiritrinum produced from Macleaya cordata Will. Is active against gram+ and gram- microorganisms.

1% Alcohol solution of Chlorophylliptum and chlorophylliptum oil. Alcohol solution should be dissolved before use. Chlorophylliptum is obtained from the leaves of Eucalyptus.

Ectericidum – antiseptic produced from fish oil. Is active against staphylococci, proteus and other microbiota.

Rp.: Sol. Chlorophyllipti spiriruosal 1% – 200 ml
D.S. 1 tablespoonful dissolve in 1L of water as mouth rinse
In the concentration 1:50 for applications.

Rp.: Ectericidi 50 ml
Sol. Novocaini 0,5% – 10 ml
M.D.S. For application and irrigation of oral cavity.

Salvinum obtained from the leaves of Salvia officinalis L. Has antiinflammatory and astriction effect on the gums. Is recommended as mouth rinse.

Romazulan – obtained from Flores Chamomillae.

Rp.: Romasulani 100,0
D.S. 1,5 table spoon dissolve in 1 l of water as mouth reinse.

Infusion and ointment of Calendulae are broadly used in periodontal therapy in the forms of applications, instillations, mouth rinse solutions.

Lysocym – the enzyme present in saliva, tears. Has bacteriolytic, antiinflammatory, mucolytic effects. Is active to gram + and some gram - microorganisms. Is used as 0,05% solution.

Rp.: Lisocymi 0,1

D.S. Powder has to be dissolved in 10 ml of isotonic solution NaCl or 0,25% solution of Novocainum. For gums applications.

Questions

1. What etiologic treatment is carried out in simple gingivitis?
2. Enumerate the main groups of antiseptics.
3. What herbal medications with antiseptic and antiinflammatory effects do you know?

7.2. Treatment of Hypertrophic Gingivitis

Treatment of the Oedematic Form of Hypertrophic Gingivitis

The treatment is started by a removal of dental deposits combined with antiseptic solution and following by antiinflammatory therapy.

Nonsteroid antiinflammatory drugs ointment of indometacini 5%, 10%, ointment of butadioni 5%, 0,1% solution of mefenaminum natrium can be used locally. Steroid antiinflammatory drugs (ointment of hydrocortisoni 0,5%) sometimes are indicated in moderate and heavy cases of gums hyperplasia.

Medications can be used in the form of application or as a part of periodontal dressing. Periodontal dressings are preferable in the case of hypertrophic gingivitis because of prolonged and stronger antiinflammatory effect on the gums.

Among other medications ointment of heparinum is used in periodontal dressings. It has anticoagulative effect and

improves processes of microcirculation in the periodontal tissues.

Sometimes in the cases of light hypertrophic gingivitis gums condition is normalized after conducting of antiinflammatory therapy. But most often after the reduction of the inflammation some hypertrophy still remains. Sclerous therapy is than used. It can be superficial and profound.

Superficial sclerous therapy is done by the application and instillation into gingival pockets of the special medications. Among them vagotil, alcoholic solutions of propolis (5-10%) (the product of apiculture), extract of the walnuts peel (juglonum), solution of rezorcinum (20-30%), plantain sap, ointment of prospidinum. Solution of Maraslavin is also widely used in the treatment of hypertrophic gingivitis. This medication is an extract of number of medicinal plants. While infiltrating in the gums, Maraslavin improves their consistence.

When the superficial sclerous therapy is not effective the profound sclerous therapy or surgical measures (gingivectomy) are indicated.

As a ***profound sclerous therapy*** intragingival injections (injections to the apex of interdental papilla) of some medications are used. Such solutions are injected in very small amounts 0,1-0,2 ml. 50-60% solutions of glucose, alcoholic solution of chlorophylliptum, cytostatic medication - solution of Novembechini (10 mg is dissolved in 10 ml of Isotonic Solution of NaCl) are used for a profound sclerous therapy. Injections are made to interdental papillas once a week and repeated 2-3 times.

In fibrous form of hypertrophic gingivitis the profound sclerous therapy or gingival surgery are indicated.

Surgical measures can be proceeded by the electrocoagulation for preventing the disease recurrence. Criosurgery and lazer techniques are also recommended.

Questions

1. What are the treatment options for the oldematic form of hypertrophic gingivitis include?
2. What medications are applied for antiinflammatory therapy?
3. What is the main purpose of sclerous therapy?
4. In what cases he surgical treatment of gingival enlargement is recommended?
5. What medications can be used for the superficial sclerous therapy?
6. What medications and how are they used to conduct the profound sclerous therapy in patients with hypertrophic gingivitis?

7.3. Treatment of Acute Necrotizing Ulcerative Gingivitis (ANUG)

The treatment of ANUG has to be complex etiotropic, pathogenic, symptomatic. It must be also local and general.

Process in the gums can be compared with the course of the wound healing in general surgery where two phases can be distinguished: ***hydratation and dehydratation with epithelization***.

In the phase of ***hydratation*** the surgical processing of the ulcerations and necrotic tissues is of great importance. It is carried out under the local applicational or infiltrational anaesthesia. For applicational anaesthesia Solution of 1% dicaini, 1% Solution of Mefeminatum Na, 4-5% Solution of propolis, 0,5-1% Novocaini or 10% Lidocaini spray (it has an irritable effect on the inflammed gums) are used.

After the anaesthesia removal of plaque and calculus is necessary. It has to be followed by antiseptic irrigations. In this case broad-spectrum antiseptics, active to gram+ and gram-

microorganisms are recommended. Among them 0,2% solution of Chlorhexidinum bigluconatis, derivatives of nitrofuran: Solution of Furacilinum (1:5000). Solution of Furaginum (1:13000). Than mechanical debridment of necrotic tissues and debris is carried out (with the help of excavator). Caries cavities have to be treated with a strong antiseptic solution and temporary filled.

To improve the effect of mechanical debridment proteolytic enzymes (trypsini, chimotrypsini, terrylitini) are used. 1 mg of proteolytic enzyme is dissolved in 1 ml of one of the solutions – 0,5% novocaini, isotonic solution of NaCl, 0,2% Solution of Chlorhexydinum Bigluconatis, solution of antibiotics (streptomycin, morphocyclin, lincomycin). Metronidazolum (in the forms of solutions and ointments), and also metronidazolum with chlorhexidine 0,2% are especially effective in the treatment of ANUG. Proteolytic enzymes can be dissolved in glycerinum or tocopherol acetatum (vit. E) (1 mg to 1 ml), but in this case their penetration into the gums will be delayed. Applications including enzymes, antibacterial medications sometimes can be connected with Dymetyl-Sulfoxidum which increases gums permeability. Applications are fixed on the gums for 10-15 minutes.

Applications of above-mentioned medications can be followed by oxygenotherapy, that is a simultaneous application of cotton rolls impregnated with KMnO4 1:10000 and 3% solution of H_2O_2. They produce intensive oxygen secretion and inactivate anaerobic microorganisms. These applications are made 2-3 times in succession.

Local treatment in the hydratation period of ANUG is connected with a general treatment. Especially general treatment is necessary in moderate and heavy stages of the disease, when the process spreads to more that 5-6 regions of the dentition.

General therapy includes desintoxication remedies: Solution of Natrii thiosulfas (Rp.; Natrii thiosulfatis 10,0 Aq. destill. 100 ml, M.D.S. 1-2 tablespoons 3 times a day), lot of natural juices, water with lemon; antihistamine medications; vitamin therapy (especially vit. C., A, B); not-irritative food reach in fruits and vegetables.

Antibacterial therapy includes metronidazol (0,25g 3 times a day within 5-7 days) or lincomicyn (0,25-0,5g 3 times a day 2 hours before or after the meal, rondomicyn (0,3g 2-3 times a day after the meal).

In the phase of ***dehytratation*** (which started in 3-5 days after the hydratation stage of ANUG) medications stimulating regeneration are very important. Among them locally (in the forms of applications and as the part of periodontal dressings) are used: vit. A, E and their combination, natural oils – Oleum Hippopheal, Oleum Rosae. Remedies stimulating metabolic processes are effective in this phase of treatment (5-10% ointment of Methyluracilum), biogenic stimulators: juices of Colanchoe, Aloe (Extractum Aloes fluidum, linimentum Aloe), Biossedum, Solcoseryl ointment, ointment "Propoceum" (includes 10% extract of propolis), aerolosolum "Proposolum", synthetic balms Vinilin, Vinizol.

Medications which are produced from the cattle cartilages and belong to the group of acid mucopolysaccharids with antiinflammatory and stimulating regeneration properties - Chonsuridum, Luronidum.

Rp.: Chonsuridi 0,1

D.t.d. N 6

S. For gums application. Before treatment dissolve 1 amp. in 10 ml of 0,5% Novocainum or isotonic solutions.

Questions

1. What phases are distinguished in the course of ANUG?

2. What is the treatment plan in the hydration phase of ANUG?
3. What antimicrobial medications are of particular importance in the treatment of ANUG?
4. What general therapy is recommended in patients with ANUG?
5. What is the treatment of the dehydratation phase of ANUG?
6. Name the medications which stimulate the regeneration in the periodontal tissues.
7. Name some medications belonging to the group of biogenic stimulators.

Test control

1. Electrosurgery, when used for periodontal surgery may be used to:

a) to remove granulation tissue interproximally
b) to remove a thin flap of tissue
c) to make incisions
d) b+a
e) none of the above

2. Periodontal dressing may contain which of the following?

a) resin
b) phelon
c) tannic acid
d) steroids
e) resin and tannic acid

3. In periodontal diseases the intrabony pocket:

a) is always present in the radiograph
b) may be absent in the radiograph
c) is always absent in the radiograph
d) is never diagnosed by means in the radiograph
e) none of the above

4. The most frequent methods of treatment of gingival enlargement associated with phenytoin hyperplasia is:

a) flap surgery
b) curettage
c) gingivectomy and gingivoplasty
d) osseous transplants
e) simple incisions

5. Healing following a gongivectomy occurs by:

a) primary intention

b) secondary intention
c) a combination of primary and secondary intention
d) none of the above
e) all of the above

6. In both, gingivitis and periodontitis which of the following changes is always sure:

a) crestal alveolar bone resorption
b) periodontal pockets
c) gingival inflammation
d) none of the above
e) all of the above

7. The most likely interpretation for the radiolucent area at the apex of pin-restored maxillary central incisors

a) the nasal fossa
b) a nasoalveolar cyst
c) a nasopalatine duct cyst
d) chronic apical periodontitis
e) the foramen of the incisive canal

8. The subgingival plaque at the base of a 7-8 mm interproximal intrabony pocket in a patient with periodontitis can effectively be disrupted by using

a) a toothpick
b) unwaxed dental floss
c) a mouthrinse containing chlorhexidine
d) the sulcular method of toothbrushing
e) none of the above

9. The destruction of which fiber group allows the junctional epithelium to migrate in an apical direction:

a) circular
b) horizontal

c) dentinogingival
d) alveologingival
e) none of the above

10. To reduce the sensitivity to thermal changes after removal of a periodontal dressing, it is the best to:
a) replane the root
b) adjust the occlusion
c) prescribe a desensitizing dentifrice
d) desensitize the roots with an appropriate medicament
e) keep the roots free of bacterial plague

11. Chronic gingivitis is caused by
a) calculus
b) occlusal trauma
c) organisms invading tissues
d) retention of food and food breakdown products
e) by products of mixed microorganisms massed on tooth and tissue surfaces

12. The most numerous cells in the inflammatory exudate of an acute periodontal abscess are:
a) neotrophils
b) eosinophils
c) basophils
d) lymphocytes
e) monocytes

13. Plaque is a major etiologic factor in which of the following conditions?
a) gingival atrophy
b) occlusal traumatism
c) desquamative gingivitis
d) juvenile periodontitis

e) none of the about

14. Which of the following structures will be most likely affected when the trauma from occlusion is present:

a) enamel
b) cementum
c) alveolar bone
d) the periodontal ligament
e) the epithehal attachment

15. The primary reason for placing a surgical dressing after a gingivectomy is to

a) to prevent hemorrhage
b) to accelerate healing
c) to stabilize the teeth
d) to retard plaque accumulation
e) none of the above

16. A periapacal cyst and a dental granuloma have all of the following histologic features in common EXCEPT

a) lymphocytes
b) plasma cells
c) fibrous connective tissue
d) an epithelial-lined lumen
e) stratified squamous epithelium

17. What is the main factor in the determination of patient's age?

a) panoramic radiography
b) profile teleradiography
c) hand radiography
d) face teleradiography
e) to ask the patient about his age

18. A disquamative gingivitis is frequently diagnosed:
a) among adolescent
b) among women over 45 years
c) at puberty
d) among pregnant women
e) none of the above

19. In the case of initial periodontal lesion with pockets depth up to 5 mm, the treatment of choice is:
a) a modified wideman flap
b) a full thickness flap
c) a partial thickness flap
d) a gingivectomy
e) none of the above

20. The treatment of ulceronecrotic gingivitis in patients without any systemic diseases includes:
a) debridement and health motivation instructions
b) antibiotictherapy (penicilline)
c) occlusal adjustment
d) gingivoplasty
e) none of the above

21. The gingiva of the pregnant woman have the tendency to:
a) an ulceration
b) the hypertrophy
c) the recession
d) an advanced parodontitis
e) none of the above

22. Which of the following is usually associated with a lateral periodontal cyst?
a) a vital tooth
b) a nonvital tooth

c) a periapical rarefaction
d) diffuse ostitis
e) an intact lamina dura

23. The width of keratinised gingiva is:
a) The distance between the free gingival groove and mucogingival junction
b) The distance between the crest of free gingiva and mucogingival junction
c) The distance from the base of gingival sulcus or periodontal pocket, along the epithelial surface over the crest of free gingiva to the mucogingival junction
d) The distance between the mucogingival junction and the projection on the external surface of the bottom of the sulcus or the periodontal pocket

24. Basal lamina:
a) Is synthesised by the connective tissue cells lying just beneath epithelium
b) Is attached to the underlying connective tissue with the help of hemidesmosomes
c) Consists of a polysaccharide-protein complex
d) Is 50-100 A wide

25. Junctional epithelium:
a) Is attached to the enamel by means of a basal lamina with lamina lucida adjacent to tooth surface
b) Undergoes decrease in number of layers as age progresses
c) Is attached to a fibrillar cementum by means of zonulae occludens
d) Has a higher turnover of epithelial cells than oral epithelium

26. The colour of attached gingiva in health, is determined by:

a) The presence of melanophores
b) Degree of keratinisation of epithelium
c) Vascular supply
d) All of the above

27. The periodontal ligament is:
a) Thinner on mesial root surface
b) Thinner on distal root surface
C. Same in thickness distally and mesially
d) Thicker on mesial surface

28. The periodontal fibres which are consistent and are reconstructed even after destruction of alveolar bone is:
a) Alveolar crest group
b) Transseptal group
c) Oblique fibres
d) Apical fibres

29. Gingival fluid does not perform one of the following functions:
a) Contains plasma proteins which may improve adhesion of the epithelial attachment to the tooth
b) Possesses antimicrobial properties
c) Exerts antibody activity in defence of gingiva
d) Provides nutrition to junctional epithelium via diffusion

30. Histologically, pregnancy related gingival enlargement shows:
a) A thinner stratified squamous epithelium
b) Endothelial cell proliferation
c) Densely arranged collagen bundles
d) Generalised acute inflammatory response

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