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DISEASES OF THE PERIODONTIUM.
ETIOLOGY. DIAGNOSIS. TREATMENT.

Lviv-2023

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CONTENS

1. PERIODONTITIS. MAIN CLINICAL SYNDROMES OF PERIODONTITIS

There are 5 main syndromes present in periodontitis:
– symptomatic gingivitis,
– periodontal pocket,
– osteopathology,
– traumatic occlusion,
– pathological changes in tooth cementum (hypercementosis, cementum demineralization and resorption).

1.1. Periodontal Pocket

A periodontal pocket is a pathologically deepened gingival sulcus; it is one of the important clinical features of periodontal disease. Progressive pocket formation leads to destruction of the supporting periodontal tissues, and loosening and exfoliation of the teeth.

Signs and Symptoms

The only reliable method of locating the periodontal pocket and determining its extent is a careful probing of the gingival margin along each tooth surface.

The following clinical signs may indicate the presence of periodontal pocket:

1. Enlarged, bluish red marginal gingiva with a “rolled” edge separated from the tooth surface.
2. A reddish blue vertical zone from the gingival margin to the attached gingiva and sometimes into the alveolar mucosa.
3. A break in the faciolingual continuity of the interdental gingiva.

4. Shiny, discoloured, and puffy gingiva associates with a exposed root surface.

5. Gingival bleeding.

6. Purulent exudate in a gingival margin, or its appearance in response to a digital pressure in the lateral aspect of the gingival margin.

7. Looseness, extrusion, and migration of teeth.

8. The development of diastemata where none had existed.

Periodontal pockets are generally painless but may give rise to the following symptoms: localized pain or a sensation of pressure after eating, which gradually diminishes; a foul taste in localized areas; a tendency to suck material from the interproximal spaces; radiating pain „deep in the bone"; a „gnawing" feellng or feeling of itching in the gums; the urge to dig a pointed instrument into the gums with relief from the resultant bleeding; compaints that food „sticks between the teeth"; the teeth „feel loose", or preference to „eat on the other side"; sensitivity to heat and cold; toothache in the absence of caries.

Classification

Periodontal pocket are classified according to morphology and their relationship to adjacent structures as follows:

Gingival pocket (relative or false)

A gingival pocket is formed by a gingival enlargement without destruction of the underlying periodontal tissues. The sulcus is deepened because of the increased bulk of the gingiva.

Periodontal pocket (absolute or true)

This is the type of pocket that occurs with destruction of the supporting periodontal tissues. Absolute pockets are of two types: 1) suprabony (supracrestal), in which the bottom of the pocket is coronal to the underlying alveolar bone, and 2) infrabony (intrabony, subcrestal, or intra-alveolar), in which

the bottom of the pocket is apical to the level of the adjacent alveolar bone. In this type the lateral pocket wall lies between the teeth surface and the alveolar bone.

Pockets of different depths and types may occur on different surfaces of the same teeth and on approximating surfaces of the same interdental space.

Pockets can also be classified according to the number of surfaces involved as follows:

Simple. One tooth surface.

Compound. Two or more tooth surfaces. The base of the pocket is in direct communication with the gingival margin along each of the involved surfaces.

Complex. This is a spiral-type pocket that originates on one tooth surface and twists around the tooth to involve one or more additional surfaces. The only communication with the gingival margin is at surface where the pocket originates. To avoid missing the compound or complex types, all pockets should be probed laterally as well as vertically.

Periodontitis is characterized by the presence of true pocket, that is, the junctional epithelium has migrated down onto the root of the tooth. The bone loss which accompanies this process may be horizontal in nature, in which case the walls of the pocket are formed of tooth and soft tissue and the pocket is a suprabony pocket. If the bone loss is vertical in nature, the pocket becomes an infra- or intrabony pocket, as one or more of the pocket walls are composed of bone immediately beneath the soft tissue lining of the pocket and coronal to the base of the pocket.

One clinical sign which is always found in chronic periodontitis is that of bleeding on gentle probing of the pocket. In addition a discharge may be present and observed on probing or on applying finger pressure to the gingiva overlying the pocket.

1.2. Traumatic Occlusion

The term ***occlusion*** refers to the contact relationships of the teeth resulting from neuromuscular control of the masticatory system (musculature, temporomandibular joints, mandible, and periodontium). In the functional sense, normality or abnormality of an individual occlusion is determined by the manner in which an occlusion functions and by its effect on the periodontium, musculature, and temporomandibular joints, rather than by the alignment of the teeth in each arch and the static relationship of the arches to each other.

Three classes of functional occlusion have been identified:

Physiologic occlusion is an occlusion that exists in an individual who has no signs of occlusion-related pathosis. This implies a range of morphologic variability in the occlusion of the teeth and a sense of psychological and physical comfort. In fact, no occlusion that exists in a given mouth free of disease and dysfunction can be considered abnormal.

Nonphysiologic occlusion is an occlusion judged to be associated with traumatic lesions or disturbances in the supporting structures of the teeth, muscles, and temporomandibular joints. The criterion that determines whether an occlusion is nonphysiologic is whether it contributes to injury, not how the teeth occlude.

Therapeutic occlusion is an occlusion used to counteract structural interrelationships related to traumatic occlusion. The term is also used to describe an occlusal scheme employed in restoring or replacing occlusal surfaces so that minimal physiologic and anatomic adaptation is required.

Traumatic occlusion is an occlusion that produces tissue injury. Excessive occlusal forces may also disrupt the function of the masculature and cause painful spasms, injure the temporomandibular joints, or produce excessive tooth wear, but the term trauma from occlusion is generally used in connection with injury in the periodontium.

Trauma from occlusion may be acute or chronic. Acute trauma from occlusion results from an abrupt change in occlusal force, such as that produced by biting on a hard object. In addition, restorations or prosthetic appliances that interfere with or alter the direction of occlusal forces on the teeth may induce an acute trauma. The results are tooth pain, sensitivity to percussion, and increased tooth mobility. If the force is dissipated by a shift in the position of the tooth or by wearing away or correction of the restoration, the injury heals and the symptoms subside. Otherwise, periodontal injury may worsen and develop into necrosis accompanied by a periodontal abscess or persists as a chronic condition.

Chronic trauma from occlusion most often develops from gradual changes in occlusion. It is more common than the acute form.

Trauma from occlusion may be caused by alterations in occlusal forces and/ or reduced capacity of the periodontium to withstand occlusal forces. When trauma from occlusion is the result of alterations in occlusal forces, it is called the primary trauma from occlusion. Examples are: the insertion of a “high filling” or a prosthetic replacement that creates excessive forces on abutment and antagonistic teeth, extrusion of teeth into spaces created by unreplaced missing teeth, etc. When traumatic occlusion results from reduced ability of the tissues to resist the occlusal forces, it is called the secondary trauma from occlusion. In this case the adaptive capacity of the tissues to withstand occlusal forces is impaired by bone loss resulting from marginal inflammation. The periodontium becomes more

vulnerable to injury, and previously well-tolerated occlusal forces become traumatic.

Tissue response to increased occlusal forces occurs in three stages: injury, repair, and adaptive remodeling of the periodontium.

Under the forces of occlusion, a tooth rotates around afulcrum or axis of rotation. This creates areas of pressure and tension on opposite sides of the fulcrum. Excessive pressure produces compression of the fibers, vascular changes in periodontal ligament, stimulates resorption of the alveolar bone, with a resultant widening of the periodontal ligament space.

Trauma from occlusion can produce radiographically detectable changes in the lamina dura (loss of the lamina dura that may be noted in apices, furcations, and/or marginal areas), in the morphology of the alveolar crest, in the width of the periodontal space (especially when variations in width between the marginal area and the mid-root or between the mid-root and the apex are detected, it indicates that the tooth is being subjected to increased forces), and in the density of the surrounding cancellous bone. More advanced traumatic lesions may result in deep angular bone loss. Root resorption may also occur as a result of excessive forces on the periodontium, particularly those caused by orthodontic appliances. The radiographic changes are not pathognomic of trauma from occlusion and must be interpreted in combination with clinical findings, particularly a tooth mobility, presence of wear facets, pocket depth, and analysis of occlusal contacts and habits.

1.3. Osteopathology in Periodontitis

Osteopathology – pathological changes in the bone of alveolar process which are typical signs of periodontal disease.

Osteopathology can occur as osteoporosis, osteosclerosis, osteolysis, bone resorption and sequestrum formation (present only in osteomyelitis).

Osteoporosis – pathological process accompanied by the decrease mineralization of cancellous bone and diminishing of the bone substance in the measured unit of bone area.

Osteosclerosis is the process contrary to osteoporosis.

Osteolysis indicates to the complete bone loss occuring in the cyst and abscess formation. Bone resorption - loss of the bone of alveolar process which can be horizontal, vertical or mixed (horizontal and vertical) in nature.

Treatment of the pathological process in the alveolar bone is one of the main problems in periodontal pathology.

The bone can be considered as the system being in the constant dynamic equilibrium between the physiological resorption and formation. Resorption of the bone is achieved by osteoclasts and osteoblasts which are responsible for the formation of the new bone tissue.

Organic matrix of the bone is highly mineralized. The processes of bone remodelling are influenced by several factors. Among them nutritional factors influencing the formation of organic matrix mostly, collagen fibers: vitamins of group B, vit. C, and minerals important in the mineralization process: Ca, F, Mn, Cr, Zn etc.

The process of bone mineralization is connected with the alkaline phosphatase level in saliva, regulating the Ca/P ratio, endocrine glands activity, especially thyroid and parathyroid hormones, sexual hormones (estradiol, progesterone).

Bone destruction is induced in the inflammatory process, where activated macrophages and lymphocytes produce factors activating osteoclasts – cells responsible for the bone resorption. Stress factors by influencing T-lymphocytes are also thought to stimulate the bone destruction.

One of the most difficult problems which arises in the long-term management of cases of chronic periodontitis is associated with the involvement of the furcation areas, in particular, of the multirooted teeth in the upper jaw. The furcation areas of the lower molars also pose problems in long-term management, but because the openings to the furcation areas are sited on the buccal and lingual aspects of the teeth they do not tend to become involved at such an early stage as the the furcation areas of the upper molars and first premolars. In the upper jaw, furcation openings are positioned in interdental areas, which are more difficult to clean, and chronic periodontitis tends to progress more rapidly. In addition, in the case of the upper molars, there are three openings to the furcation area which may be involved.

Another commonly observed sign of chronic periodontitis (caused by the bone loss in periodontitis) is migration of teeth, usually away from the deepest pocket. Thus in the case of labial migration of an upper incisor, the deepest pocket is usually found on the palatal aspect. There are other possible causes of such labial migration which should be considered, for example, loss of posterior support in cases which already have a deep overbite and a potentially unstable incisor relationship. The cause of migration of the teeth in periodontal disease has been ascribed to the pressure of the chronic inflammatory tissue and the migration may be in an occlusal direction causing elongation of the clinical crown. This type of migration may result in teeth moving into a situation where premature occlusal contacts are created and patients will frequently compain of the resultant occlusal disharmony.

1.4. The Periodontal Abscess

A periodontal abscess is a localized purulent inflammation in the periodontal tissues. It is also known as a lateral or parietal abscess. ***Periodontal abscess*** formation may occur as follows:

1. Deep extension of infection from a periodontal pocket into the supporting periodontal tissues, and localization of the suppurative inflammatory process along the lateral aspect of the root.

2. Lateral extension of inflammation from the inner surface of a periodontal pocket into the connective tissue of the pocket wall. Localization of the abscess results when drainage into the pocket space is impaired.

3. In a pocket that describes a tortuous course around the root (complex pocket), a periodontal abscess may form in the cul-de-sac, the deep and of which is shut off from the surface.

4. Incomplete removal of calculus during treatment of a periodontal pocket. In this instance, the gingival wall shrinks, occluding the pocket orifice, and a periodontal abscess occurs in the sealed-off portion of the pocket.

5. A periodontal abscess may occur in the absence of periodontal disease, following trauma to the tooth or perforation of the lateral wall of the root in endodontic therapy.

Among the general reasons of the periodontal abscess the main is the decrease reactivity of the immune system.

Classification

Periodontal abscesses are classified according to location as follows:

1. Abscess in the supporting periodontal tissues along the lateral aspect of the root. In this condition, there is generally a sinus in the bone, which extends laterally from the abscess to the external surface.

2. Abscess in the soft tissue wall of a deep periodontal pocket.

Clinical features

Periodontal abscess may be acute or chronic. Acute lesions often subside but persist in the chronic state, whereas chronic lesion may exist without having been acute. Chronic lesions frequently undergo acute exacerbations.

Acute abscess. The acute periodontal abscess is accompanied by symptoms such as a throbbing radiating pain, exquisite tenderness of the gingiva to palpation, sensitivity of the tooth to percussion, tooth mobility, lymphadenitis, and systemic effects such as fever, leukocytosis, and malaise.

The acute periodontal abscess appears as an ovoid elevation of the gingiva along the lateral aspect of the root. The gingiva is oedematous and red, with a smooth, shiny surface. The shape and consistency of the elevated area vary. It may be dome-like and relatively firm, or pointed and soft. In most instances, pus may be expressed from the gingival margin by a gentle digital pressure. Occasionally, the patient may present symptoms of an acute periodontal abscess without any notable clinical lesion or radiographic changes.

Chronic abscess. The chronic periodontal abscess usually presents a sinus that opens into the gingival mucosa somewhere along the length of the root. There may be a history of an intermittent exudation. The orifice of the sinus may appear as a difficult-to-detect pinpoint opening, which when probed reveals a sinus tract deep in the periodontium. The sinus may be covered by a small, pink, beadlike mass of granulation tissue.

The chronic periodontal abscess is usually asymptomatic. The patient may report episodes characterized by a dull gnawing pain, slight elevation of the tooth, and a desire to bite down and grind the tooth. The chronic

periodontal abscess often undergoes acute exacerbations with all the associated symptoms.

Radiographic appearance

The typical radiographic appearance of the periodontal abscess is that of a discrete area of radiolucence along the lateral aspect of the root. However, the radiographic picture is not always typical because of many variables such as:

1. The stage of the lesion. In the early stages the acute periodontal abscess is extremely painful but presents no radiographic changes.

2. The extent of bone destruction and the morphology of the bone.

3. The location of the abscess.

Lesions in the soft tissue wall of a periodontal pocket are less likely to produce radiographic changes than those deep in the supporting tissues.

Abscesses on the facial or lingual surface are obscured by the radiopacity of the root; interproximal lesion are more likely to be visualized radiographically.

The radiograph alone cannot be relied upon for the diagnosis of a periodontal abscess.

Treatment of the Periodontal Abscess

The purpose of treatment of an acute abscess is to alleviate pain, control the spread of infection and establish a drainage. Drainage can be established through the pocket or by means of an incision from the outer surface.

Drainage through the pocket is started after application of a topical anaesthetic by a flat instrument, which is introduced to the pocket in an attempt to distend the pocket wall. When the drainage can not be established via the pocket or when the abscess can be seen pointing through the gingiva, an external incision is indicated. The abscess has to be gently

palpated to locate the most fluctuant area, through which a vertical incision is made, extending from the mucogingival fold to the gingival margin. The blade should penetrate to a firm tissue to be sure of reaching deep, purulent areas. After the initial extravasation of blood and pus, the clinician shoud irrigate the area with warm water, antiseptic solution (Chlorhexidinum Bigluconatis 0,2%). If the tooth is extruded, it should be ground slightly to avoid contact with antagonists. Patient is instructed to rinse thoroughly with a solution of a teaspoon of salt in a glass of warm water and to return the next day. In addition to the rinses, antibiotics are prescribed for patients with fever. Analgesics are prescribed for pain.

The chronic Periodontal Abscess is treated by Flap Operation (see chapter 9.2.)

Questions

1. Give the definition of the periodontal abscess.
2. What types of periodontal abscesses do you know?
3. What is the etiology of the periodontal abscess?
4. What are clinical signs of the periodontal abscess?
5. Comment on the radiographic appearance of the periodontal abscess?
6. How is the periodontal abscess treated?

1.5. Pathological changes in tooth cementum

Root cementum as a specialized mineralized tissue plays an important role in connection of periodontal fibers and retaining the tooth in the alveolar. The level of cementum mineralization influences the condition of the periodontium. One of the possible reasons for a periodontal pocket formation,

is known to be weakening of the cementum deposition, and thus reduction of the barrier for the epithelium migration.

Mineralization of the cementum in the superficial and deep layers is different. In periodontitis, the level of Ca and P is known to be higher in roots exposed in periodontal pockets and their microhardness changed. This process is known as hypercementosis. Besides hypercementosis, cementum in periodontitis teeth is often dystrophic and hypomineralized, that leads to the formation of carious cavities in tooth cementum.

Results of microprobe radiospectral analysis of Ca and P content in the coronal part of root cementum

Characteristic of examined pattern	Content of Ca (%)	Content of P (%)
1. Cementum free of calculus	21,7	4,0
2. Cementum covered with calculus	20,6	3,7
3. Caries cementum	16,2	2,8
4. Polished cementum	23,4	7,9

Analysis error ± 1,0%

After the hand and even ultrasound scaling some changed and contaminated cementum is remained which is the source of intoxication to periodontal tissues. Additional polishing of the cementum after scaling made the surface smoother and less contaminated. Polished and nonpolished cementum differes significantly ($p<0,01\%$) in microhardness (Cementum microhardness was estimated at a depth of 25-50□m by Viker"s method). Microhardness of nonpolished cementum was lower ($99,5\pm1,4 kg/mm^2$) than that of the polished cementum ($109,8\pm2,2 kg/mm^2$). Obtained clean and

dense cementum structure is probably optimal for rehabilitation and preservation of dento-gingival structure.

1.6. Clinical signs of Different Stages of Periodontitis

I-st stage of Periodontitis

Patients' complaints are: bleeding, sometimes a foul taste and the increased sensibility of teeth to cold and hot irritants (because of tooth cementum exposure and demineralization). Periodontal pockets are up to 3,5-4 mm, subgingival calculus is always present and revealed by probing. X-ray indicates osteoporosis of the crests of the interdental bone and destruction of lamina dura. Bone destruction is determined up to 1/3 of the tooth roots length. Horizontal bone loss prevails but the vertical bone destruction can be found in localized areas. In the presence of acute inflammation the first stage of teeth mobility can be observed (that is vestibulo-lingual mobility).

II-nd stage of Periodontitis

Chief complaints are: teeth mobility, changing of the position of some teeth, often fan-like divergence of the teeth. However, it should be stressed that mobility is not a reliable indicator of disease as it may have arisen due to excess loading of the tooth or normal forces acting upon a reduced but healthy periodontium, for example following treatment of periodontal disease, apicectomy or just in the case of a tooth with a short root.

Sometimes bleeding persists sometimes it is not intensive but often increases in the periods of exacerbations. Chewing is often painful. Foul taste almost always accompanies this stage of periodontitis.

Periodontal pockets are up to 4-6 mm. Vertical bone loss prevails the horizontal one, that means the presence of

infrabony pocket and increase amount of pathogenic, anaerobic, microorganisms in the periodontal pockets. Bone destruction up to 1/2 of teeth roots. Exacerbations episodes with periodontal abscesses formation are often findings in the II-nd stage of Periodontitis . Loss of the 50% of the alveolar bone leads to the secondary traumatic occlusion (1^{st}-2^{nd} stages of teeth mobility, divergence and changing position of the teeth).

Degeneration processes are seen in gingival tissues - changing of the gingival contour and gums recession. Cementum caries is typical for the advanced periodontitis.

The III-rd stage of periodontitis

Chief complaints are pain during mastication, esthetic complaints on the increased teeth crowns (accoding to the recession of the gums) and expressed teeth mobility. Gingival tissues are totally changed, bluish and oedematous. Periodontal pocket – 6-7 mm and more. Pronounced traumatic occlusion with II-III stages of teeth mobility. Bone resorption occurs to be more than 2/3 length of the teeth roots. Bone destruction is mostly of vertical nature. Tooth cementum presents areas of destruction and caries. Retrograde pulpitis and endoperiodontal lesions are always present in this stage of periodontitis.

Questions

1. What are the main clinical syndromes of periodontitis?
2. What types of periodontal pockets do you know?
3. What are the clinical signs of a periodontal pocket?
4. How can the syndrome of osteopathology in periodontitis be characterized?
5. What are the reasons of teeth migration in periodontitis?
6. Clinical signs of different stages of periodontitis.

1.7. The Endo-, Perio- Lesions

Periodontology and endodontology are often considered as two separate entities yet clinically they are closely related and this must influence our diagnosis and treatment. Communication between the periodontal and pulp tissues may be via the apical foramina, lateral canals, root fractures or perforations. Lateral canals should be considered channels of communication which are capable of allowing noxious material of microorganisms to pass in either direction. Endo Perio lesions can be classfied as (Messing, Stock, 1988):

Class 1 – Primary endodontic lesion draining through the periodontal ligament;

Class 2 – Primary endodontic lesion with secondary periodontal involvement;

Class 3 – Primary periodontal lesions;

Class 4 – Primary periodontal lesion with secondary endodontic involvement.

Class 1 – Primary endodontic lesion draining through the periodontal ligament are present as an isolated periodontal pocket or swelling on the side of the tooth. The patient rarely complains of pain although there will usually be a hisrory of an acute pain episode. The lesions are caused by a necrotic pulp draining through the periodontal ligament. The furcation area of both premolar and molar teeth may be involved. These lesions should be suspected when crestal bone levels on the mesial and distal aspects of the tooth appear normal and only the furcation area is radiolucent. The prognosis following root treatment is good.

Class 2 – Primary endodontic lesions with secondary periodontal involvement occur if left untreated the primary periodontic lesion. Endodontic treatment will heal a part of lesion but complete repair will involve periodontal therapy. The prognosis is generally good.

Class 3 – Primary periodontal lesions are caused by periodontal disease. Periodontitis slowly advances down the root surface until the apex is reached. The pulp will be vital in the early stages but may become affected as the disease progresses. The tooth will become mobile as the attachment apparatus and surrounding bone are destroyed leaving deep periodontal pocketing. The periodontal involvement will not be confined in general to one tooth, except in the case of a palatal developmental groove. The prognosis for primary periodontal lesions becomes worse as the disease advances.

Class 4 – Primary periodontal lesions with secondary endodontic involvement occur if the pulp of periodontally involved teeth become inflamed. The periodontal procedure of root planing may interfere with the blood supply to part of the pulp through lateral canals. Root treatment of terminal cases of periodontal disease which have not responded to periodontal therapy is recommended. The prognosis for these lesions is poor.

It has been discovered (Jansson et al., 1995) that an intra-canal infection of endodontic pathogens stimulates epithelial downgrowth along denuded dentin surface with a marginal communication. Extrapolated to the clinical situation, endodontic infections in periodontal-prone patients may augment periodontitis propagation. It has been found that periodontitis-prone teeth run a significantly higher risk of losing a periodontal attachment if a root canal infection is also present and evident as a periapical radiolucency.

In all forms of generalized periodontitis the periapical changes in the alveolar bone can occur as granulating or granulomatous destruction. Frequency of these two processes, however, is different in the periodontitis of different stages of heaviness. In the periodontitis of the I-st stage of heaviness granulating periapical destruction occurs in 37% of patients while granulomatous destruction – in 62% of patients. In

periodontitis of II-nd and III-rd stages of heaviness, on the contrary, a granulating periapical bone destruction has been observed more often, in 77% of patients (Deneha, 1995).

In the immunological investigations of the periodontal blood it has been found that the state of local immunity differs considerably in patients with generalized periodontitis when compared to healthy individuals. In the foci of endo-, perio-lesions immunological changes have been strongly expressed than in the regions of exclusively periodontal lesions. It has been revealed (Deneha, 1995) that in regions of endo-, perio-lesions with a granulomatous destruction in the periapical bone (periapical cyst granuloma, periapical cyst) T-helper/T-suppressor index is increased, while in patients with the granulating type of destruction this index is decreased when compared to the results in solely periodontal lesions. The regions of endo perio lesions, therefore, can be considered the weak points in the development of the periodontal destructions. Because the progression of the periodontal breakdown is characterized by the decrease of T-helper/T-suppressor index it can be also concluded that the presence of numerous foci of periapical granulating bone destruction in periodontitis patients is one of the sign of the rapidly progressing periodontal process. The fact of prevailing of granulomatous type of periapical destruction testifies obviously to the slowly progressing periodontal disease (Deneha, 1995).

1.8. New Approach in the Estimation of the Nature of Generalized

Periodontitis

According to the peculiarities of clinical status and rate of progression of pathological changes in periodontal tissues and immunological parameters in the peripheral and

periodontal blood Deneha I. (2000) indicates to the existence of the three forms in the course of periodontitis.

1. Slowly progressing not active periodontitis.
2. Rapidly progressing aggressive periodontitis.
3. Rapidly progressing dystrophic periodontitis.

Progressing of the periodontitis has been considered not active in the case of interdental alveolar bone loss and additional loss of the attachment level being up to 1-2 mm during the year. In aggressive course of periodontitis the above-mentioned parameters are equal or higher than 3 mm.

Slowly progressing not active periodontitis is characterized by rare exacerbations of periodontal process (not more than 1-2 exacerbations in a year) and protracted course (10-12 years in case of moderate stage of periodontitis) (fig.26,27,28,29,40,41). Moderate activation of T-cell and local humoral immunity is present in patients with the slowly progressing not active periodontitis.

Rapidly progressing aggressive periodontitis is characterized by the high level of indices representing an inflammation in the periodontal tissues (bleeding index, index of gingivitis, test of capillaries resistance to vacuum) as well as indices of the inflammatory-destructive periodontal process activity (the frequency of exacerbations of the periodontitis, the predominance of vertical resorption of interdental alveolar bone, the percentage of teeth with deep periodontal pockets and clinically active periodontitis, periodontal index).

When compared with two other forms of disease, especially with a slowly progressing not active periodontitis, in patients with a rapidly progressing *active* periodontitis periodontal inflammation is strongly pronounced and the activity of periodontal process is more expressed. These changes indicates to the aggressive cause of generalized periodontitis in this group of patients.

Rapidly progressing periodontitis is characterized by active and massive bone loss. This form of periodontitis is difficult in diagnosis and treatment. Numerous immunological investigations indicate to the changes of T-cell subsets in general and local level in patients with a rapidly progressing periodontitis. Immunological changes local as well as general, in this form of generalized periodontitis, are much more activated and overstrained when compared with a slowly progressing not active periodontitis. Level of immunological parameters in rapidly progressing periodontitis are also much more subjected to clinical changes. Rapidly progressing periodontitis is characterized by often abscess formation (3-4 times a year), intensive bleeding of the gums, teeth mobility. In rapidly progressing process periodontal disease of moderate (II-nd) stage of heaviness proceeds for 5-7 years with final complete teeth loss without treatment.

Rapidly progressing dystrophic periodontitis is characterized by the pronounced recession of the gums, existence of the foci of osteosclerosis in the bone of interdental septa simultaneously with the vertical bone resorption and pocket formation.

Standard treatment of the aggressive forms of periodontitis is often not so much effective as in slowly progressing periodontitis. Some investigators in this case consider these forms of periodontitis as refractory periodontitis.

Qualitative change of obligate and facultative species of large intestinal microbiota as well as conventionally pathogenic microbiota have been revealed in 88,3% of patients with generalized periodontitis. Clear correlation between the changes in systemic cellular immunity and the degree of dysbacteriosis in the colon has been revealed. Traditional complex treatment of generalized periodontitis didn’t lead to the normalization of microbiota in large intestine, but the intensity of disbiotic changes was lower in treated patients.

Sufficient degree of the normalization of immunological indices after the conducted traditional treatment have been observed only in the patients with normal eubiotic condition of the colon.

Thus to achieve optimal immunorehabilitational effects, leading to the stable disease remission in the complex treatment of the patients with generalized periodontitis it is very important to remove dysbacteriosis of large intestine. Specific immunomodulative therapy is also especially indicated to the patients with a rapidly progressing periodontitis.

2. TREATMENT OF PERIODONTITIS

2.1. General Principles of the Complex teatment of Periodontitis

Treatment of periodontitis has to be complex, that means including the therapeutical, surgical, prosthetic and orthodontic elements and physiotherapy, mostly in the period of maintenance care.

Treatment of periodontitis always includes general and local elements. It can be etiological (hygiene measures, removal of the unsatisfactory prosthetic appliances, antimicrobial therapy) and pathogenic (antiinflammatory drugs, vitamins, enzyme therapy, etc).

Therapeutical (or conservative) treatment is aimed at the reduction of symptomatic gingivitis. Broad spectrum of medications are used at this stage of treatment - antiinflammatory drugs, antimicrobial medications, proteolytic enzymes, enzymes inhibitors, biogenic stimulators sometimes immunomodulators. Medications are applied locally, mostly in

the forms of periodontal dressings sometimes as applications and instillations or in the form of electrophoresis. General treatment is always recommended in periodontitis patients. It includes diet normalization, antiinflammatory drugs, vitamin therapy, immunomodulators, antibiotic therapy.

Antiinflammatory therapy in the treatment of periodontitis

The existence of an inflammatory component of periodontal disease is well established. The systemic and topical use of antiinflammatory drugs in the treatment of gingivitis and periodontitis has been practised for many years. Steroids – as a group – were found to inhibit an enzyme (phospholipase A2) which releases arachidonic acid from lipid membranes. Arachidonic acid is subsequently oxidized by cyclooxygenases or lipoxygenases (arachidonic acid cascade) to a series of inflammogenic products, such as prostaglandins and leukotrienes. Since arachidonic acid is an unsaturated C-20 (eicosanoic) acid, the oxidized derivates are today known as eicosanoids.

Because of numerous side effects steroids are not widely used in the antiinflammatory treatment of periodontal diseases.

NSAIDs (non-steroidal antiinflammatory drugs) function differently from steroids. They do not inhibit release of arachidonic acid but interfere with the subsequent oxidative processes, thereby reducing (or eliminating) the generation of inflammogenic eicosanoids.

One of the most important aspects of the use of NSAIDs is their influence on the destructive effect of periodontal disease on bone loss. Any reduction of inflammation as measured by a gingival index, crevicular fluid or a bleeding index does not necessarily correlate with the bone loss.

The most widely used NSAIDs, such as aspirin, indometacin or ibuprofen, interfere with the cyclooxygenase pathway which leads from arachidonic acid to the prostaglandins. Some prostaglandins reduce leukocyte chemotaxis, while prostaglandin F (PGF) enhances chemotaxis. The related compounds prostacyclin (PGI2) and thromboxane (TXA2) are also antagonistic to each other, the former is a potent vasodilator, while the latter acts as a vasoconstrictor.

The effect of NSAIDs can be also associated with their specific effects in reducing the vascularity and permeability of small blood vessels (Heasman and Seymour, 1990). It has been also shown that NSAIDs (indometacin especially, used locally as 5% ointment in periodontal dressings reduces the content of C-reactive protein in the periodontal foci of inflammation, which is considered the marker of the inflammatory process activity (Deneha, 2000).

Commonly-used dental medicaments are also known to inhibit the formation of prostaglandin. Eugenol, which is one of the most potent inhibitors, has been used by dentists for many years to alleviate alveolitis and endodontic inflammation. Similarly, guaiacol and thymol have been widely used for similar purposes.

Steroids and nonsteroidal antiinflammatory drugs (NSAIDs), in spite of the large number of available drugs treatment of periodontitis, can’t be confined to the conservative measures as it was in the case of independent gingivitis. The main reason of all nonsafisfactory results in the conservative treatment of the periodontitis is the existence of the periodontal pockets which can not be eliminated with the help of medications. The only way for pockets elimination is a periodontal surgery.

2.2. Periodontal Sugery

Periodontal surgery includes: closed and open curettage, gingivectomy, periodontal flap operations. As symptomatic measures gingivotomy (that is horizontal or vertical incisions) is used mostly in the case of abscess formation. Another surgical procedures are those of mucogingival surgery: deepening of the vestibulum oris, frenulectomy, lateral and vertical repositions of the periodontal flaps.

Pocket elimination by scaling and curettage.

As a guide to treatment, periodontal pockets may be subdivided into three critical zones (Garranza F.A.,1979).

Critical Zones in Pocket Elimination.

Zone 1. The soft tissue wall and junctional epithelium.

The soft tissue wall of the pocket is inflamed and presents varying degrees of degeneration and ulceration with engorged blood vessels close to the surface, often separated from the contents of the pocket by only a thin layer of tissue debris. In this zone should be determined the following aspects:

Whether the pocket wall extends in a straight line from the gingival margin or follows a tortuous course around the tooth.

The number of tooth surfaces involved by the pocket.

The location of the bottom of the pocket on the tooth surface, and the pocket depth.

The relationship of the pocket wall to the alveolar bone. Is the entire pocket coronal to the crest of the bone (suprabony pocket), or is there bone lateral to the pocket wall (infrabony pocket)?

Zone 2. The tooth surface.

Adherent to the tooth are calculus and other tooth surface deposits of varying amounts and texture. The superficial calculus is generally clay-like in consistency,

obvious, and easily detached by a well-directed instrumentation. However, deep in the pocket the calculus is hard, flint-like, and tenaciously adherent to the surface. In the coronal portion of the root the cementum is extremely thin, and a lodge is often formed at the cemento-enamel junction, which must be taken into consideration when the tooth is scaled. The cementum surface may be softened by caries. It may be deformed by adherent cementicles.

The pocket itself contains bacteria, bacterial products, the products of food decomposition, and calculus, all bathed in a slimy mucous medium. Pus may or may not be present.

In this zone the following has to be determined:

1. The extent and location of deposits.
2. The condition of the tooth surface; the presence of softened, eroded areas.
3. The accessibility of the root surface to the necessary instrumentation.

Zone 3. The connective tissue between the pocket wall and the bone.

In this zone the operator should determine whether the connective tissue is soft and friable, or firm and bound to the bone. This is a significant consideration in the treatment of infrabony pockets.

Pocket elimination should be systematic, beginning in one section and proceeding in orderly sequence until the entire mouth is treated. Treatment is usually started in the right maxillary molar area, unless it is urgently needed in another section. The number of teeth included at each visit varies with the skill of the operator, the type of a patient, and the severity of periodontal involvement.

Closed curettage.

Curettage is employed to remove the diseased inner lining of the pocket wall, including the junctional epithelium. If the junctional epithelium is permitted to remain, the epithelium

from the crest of the gingiva will proliferate along the curetted pocket wall to join it and prevent any possibility of reattachment of the connective tissue to the root surface. Curettes can be used for this purpose with cutting edges on two sides of the blade so that the root is smoothed in the same operation.

Curettage removes a degenerated tissue, proliferating epithelial buds, and granulation tissue, which will go to form the inner aspect of the pocket wall, and creates a cut, bleeding connective tissue surface. The bleeding causes shrinkage in the height of the gingiva and reduction in a pocket depth and facilitates a healing by removing tissue debris.

Step 1. Isolation and anesthetization of the area.

Step 2. Removal of the supragingival calculus.

Step 3. Removal of the subgingival calculus.

A curette is inserted to the bottom of the pocket just beneath the lower border of the calculus and the calculus dislodged.

Step 4. Planning of the tooth surface.

Step 5. Curettage of the soft tissue wall.

Step 6. Polishing of the tooth surface.

Root surfaces are polished by using a rubber cups with pastes. The flexibility of the rubber cup permits access to the subgingival area without traumatizing the tissues. Brushes are not used for polishing the root surfaces at this stage because of the difficulty of avoiding soft tissue injury.

Healing following scaling and curettage.

Immediately after scaling and curettage, a blood clot fills the gingival sulcus. This is followed by a rapid proliferation of granulation tissue with a decrease in the number of small blood vessels as the tissue matures. Restoration and epithelization of the sulcus generally require from two to seven days, and restoration of the junctional

epithelium occurs in animals as early as five days. Immature collagen fibers appear within 21 days after treatment.

Operating areas should be closed by a periodontal packing. Closed curettage is indicated in the depth of periodontal pockets 3-4 mm, that is in the I stage of periodontitis. Contraindications for this procedure are as follows: pronounced inflammation, extremely thin or fibrotic gingival wall of the pocket, ulcerations of the gingival tissues.

Open curettage.

Open curettage is done in similar steps as closed curretage with one difference that gingival wall has to be separated from the tooth surfaces and the operating field becomes visualized. Sometimes sutures are put to close the operating region but in most cases the region is protected only by the periodontal packing.

The Gingivectomy Technique.

In a limited literal sense, the term gingivectomy means excision of the gingiva. In reality, it is a two-stage operation consisting of the removal of diseased gingiva and the scaling and planning of the root surface.

Gingivectomy derives its effectiveness from the following:

1. By removing the diseased pocket wall which obscures the tooth surface, it provides the visibility and accessibility that are essential for the complete removal of irritating surface deposits and thorough smoothing of the roots.

2. By removing the diseased tissue and local irritants, it creates a favourable environment for gingival healing and the restoration of physiological gingival contour.

Indications and contraindications.

The gingivectomy technique is indicated in the following cases. Elimination of deep suprabony pockets in

which the deposits on the root cannot be seen in their entirety when the pocket wall is deflected with a probe or blast of warm air. In deep or inaccessible pockets, calculus cannot be completely removed with any degree of predictability by hand scalers if the operator must rely solely upon tactile sensation. Elimination of all suprabony pockets, regardless of their depth, if the pocket wall is fibrous and firm. Because fibrous gingival tissue does not shrink after scaling and curettage, some form of surgical treatment is necessary to eliminate the pocket. Elimination of gingival enlargements. Elimination of suprabony periodontal abscesses. Other techniques are also available for some of the above-mentioned indications.

The following two findings will contraindicate the gingivectomy technique:

a) The need for bone surgery or even for examination of the bone shape and morphology.

b) The location of the bottom of the pocket apical to the mucogingival junction.

When used for the purposes for which it is intended, the gingivectomy technique is a most effective form of treatment.

The Periodontal Flap.

A periodontal flap is a section of the gingiva and/or mucosa surgically separated from the underlying tissues to provide visibility and access to the bone and root surface. The flap also allows the gingiva to be positioned in a different location in cases of mucogingival involvement.

The basic steps for the flap technique were described early in the 20th century by several clinicians; with some modifications and refinements they constitute the techniques utilized today. The flap operation is used in many different situations and varies according to the degree of flap reflection, the amount of tissues reflected, the types of incisions utilized, and the final position of the flap.

Classification of flaps.

Periodontal flaps are classified as either full thickness (mucoperiosteal) or partial thickness (mucosal) flaps. In full thickness flaps, all of the soft tissue, including the periosteum, is reflected to expose the underlying bone. This complete exposure and access to the underlying bone is indicated if osseous surgery is contemplated. The full thickness flap is reflected by means of a blunt dissection. A periosteal elevator is used to separate the mucoperiosteum from the bone by moving it mesially, distally, and apically until the desired reflection is accomplished.

The partial thickness flap includes only the epithelium and a layer of the underlying connective tissue. The bone remains covered by a layer of the connective tissue including the periosteum. Sharp dissection is necessary to reflect a partial thickness flap. A surgical scalpel is utilized to separate the flap carefully. The partial thickness flap is indicated when the flap is to be positioned apically or when the operator does not desire to expose bone.

Surgical curettage (Modified Widman Flap)

Indication for this procedure is the periodontitis or the II-nd and the III-rd stages of heaviness with infrabony pockets.

The description of the procedure.

1. The initial incision is a scalloped internal bevel incision to the alveolar crest and 1 to 2 mm, away from the gingival margin. Care should be taken to insert the blade in such a way that the papilla is left with similar thickness as the remaining facial flap. Vertical relaxing incisions are usually not needed.
2. The gingiva is reflected with a periosteal elevator.
3. A crevicular incision is made from the bottom of the pocket to the bone, circumscribing the triangular wedge of tissue containing the pocket wall.

4. A third incision is made in the interdental spaces coronal to the bone with a curette or an interproximal knife, and the gingival collar is removed.

5. Tissue tags and granulation tissue are removed with a curette. The root surfaces are checked and scaled and planed if needed. Residual periodontal fibers attached to the tooth surface should not be disturbed.

6. Bone architecture is not corrected except when it prevents good tissue adaptation to the necks of the teeth. Every effort is made to adapt the facial and lingual interproximal tissue adjacent to each other so that no interproximal bone remains exposed at the time of suturing. The flaps may be thinned to allow for close adaptation of the gingiva around the entire circumference of the tooth and to each other interproximally.

7. Interrupted direct sutures are placed in each interdental space and sometimes covered with periodontal surgical pack.

2.3. Role of Bone Substitutes in Periodontal Therapy

Conventional periodontal treatments, such as root planning, gingival curettage, and scaling are highly effective at repairing disease-related defects and halting the progression of periodontitis. However, the conventional therapies do relatively little to prompt the regeneration of lost periodontal support structure (Acton J.G., Greenstein G., 1994). Some studies indicate that they typically result in the development of a long junctional epithelium between the root surface and gingival connective tissue rather than the regrowth of tissue that restores the architecture and function (Rosenberg E., Rose L.F., 1998).

Bone grafting is the most common form of regenerative therapy today and is usually essential for restoring all types of

periodontal supporting tissues. Histologic evidence in humans indicates that bone grafting is the only treatment that leads to regeneration of the bone, cementum, and a functionally oriented new periodontal ligament coronal to the base of previous osseous defect (Bowers G.M., Chadroff B., Carnevale R., et al, 1989, Mellonig J.T., 1991).

Periodontal regeneration, defined as the reproduction or reconstitution of a lost or injured part (The American Academy of Periodontology: Glossary of Periodontal Terms, 1992), refers to the complete restoration of functional supporting tissues, including the alveolar bone, the cementum and the periodontal ligament. Regenerative therapy refers to various modalities, such as bone grafts, root conditioning and guided tissue regeneration, that promote the body's natural ability to replace these lost periodontal support structures. Periodontal repair refers to the healing of a periodontal wound with a tissue that restores continuity but does not fully restore the architecture and function of the support structures.

New attachment – is the reunion of connective tissue with a root surface that has been deprived of its periodontal ligament. The new attachment occurs by the formation of new cementum with inserting collagen fibers. Reattachment is the reunion of connective tissue with a root surface on which viable periodontal tissue is present. Nothing new is formed.

Biology of bone healing

Bone heals in a unique way compared with other connective tissues. Rather than developing scar tissue, it has the ability to regenerate itself completely. The bone repair process begins with an inflammatory response that prompts granulation tissue to proliferate in the wound site. This granulation tissue brings in capillaries, fibroblasts, and osteoprogenitor cells. Osteoblasts are produced by the osteoprogenitor cells in the granlation tissue, and they begin to make the organic matrix of woven bone and to initiate

mineralization. This healing mass of new tissue is called the callus. Over time, it is replaced by a lamellar bone.

Osteogenesis, or the process of bone formation, begins with either osteoblasts in the patient's natural bone or from the surviving cells in the bone graft that is placed. Through a gradual healing process that begins with inflammation, bone grafts are incorporated into the patient's natural oral bone structure over time (Goldberg V.M., Stevenson S., 1992, Schenk R.K., 1994).

Several types of bone grafts, used in modern periodontology are: autograft (intraoral, extraoral), allograft (freeze-dried, fresh), xenogenic, alloplastic.

Autogenous grafts, which are harvested from the patient's own body, are considered the gold standard among graft materials because they are at retaining cell viability. They contain live osteoblasts and osteoprogenitor stem cells and heal by osteogenesis. Autogenous grafts avoid the potential problems of histocompatibility differences and the risk of disease transfer (Brunsvold M.A. Mellonig J.T., 1993). Autogenous bone can often be harvested from intraoral sites, including edentulous ridges, tori, the maxillary tuberosity, or healing bony wound or extraction sites or from extraoral sites, such as the iliac crest.

Allogeneic periodontal bone grafts (allografts) were developed as an alternative to autogenous grafts. Allografts are bone taken from one human for transplantation to another. There are various types of allografts available, including freeze-dried bone allograft (FDBA) and demineralized freeze-dried bone allograft (DFDBA). FDBA, which is not demineralized, works primarily through osteoconduction, a process in which the graft does not activate bone growth but instead acts like a scaffold for the patient's own natural bone to grow onto and within. Over time, the graft is resorbed and replaced by a new bone (Boyne P.J., 1993) and this

regenerative process is thought to be induced by a bone morphogenic protein (BMP) and perhaps other growth factors released from the allograft (Spampata R., Werther J.R., Hauuschka P.V., 1992).

DFDBA provides more bone fill than FDBA (Mellonig J.T., Bowers G.M., 1990). Removing the bone mineral appears to be a crucial factor. This process exposes BMPs or other proteins in the graft material that stimulate the formation of a new bone by osteoinduction. Human clinical studies have shown DFDBA grafts result in 2,5 to 3 mm of bone fill, which is somewhat less than autogenous bone (Garrett S.: Periodontal regeneration around natural teeth: Tooth/defect factors. In Genco R.J., Newman M.G. 1996). Although human histologic evidence indicates that DFDBA can promote the formation of new attachment apparatus on root surfaces (Bowers G.M., Chadroff B., Carnevale R., et al, 1993), some researchers and clinicians have begun to question the usefulness of DFDBA. Contrary to clinical and histologic studies that have found good regenerative and new attachment results with DFDBA (Bowers G.M., Chadroff B., Carnevale R., et al; McClain P.K., Schallhorn R.G., 1993) other studies show less predictable and even disappointing results with the material (Becker W., Schenk R., Higuchi K., et al, 1995; Pinholt E.M., Haanaes H.R., Roervik M.,et al, 1992).

Xenografts are grafts between different species. Currently, there are two available sources of xenografts used as bone substitutes in clinical practice: bovine bone and natural coral. Both are biocompatible and structurally similar to human bone and almost entirely free of the risk of disease transmission. One of the advantage of the product as a bone substitute is that it is natural. Anorganic bovine bone is the hydroxyapatite (HA) skeleton, which retains a highly porous structure similar to cancellous bone that remains after chemical or low heat extraction of the organic component. Examples of

xenografts are: Osteograf/N (CeraMed, Lakewood, CO) and BioOss (Osteohealth Co, Shirley, NY). Both have been reported to have good tissue acceptance with natural osteotrophic properties (Callan D.P., Rohrer M.D., 1993; Cohen R.E., Mullarky R.H., Noble B., et al, 1994). Histologically, no fibrous tissue or space between the HA and newly formed bone is found (Callan D.P., Rohrer M.D,1993). This is in contrast to histologic reports obtained with synthetic HA.

Alloplastic (synthetic) bone grafts are Biocoral, HTR, bioceramics, bioactive glasses. Biocora (Inoteb, Saint Gonnery, France) is calcium carbonate obtained from a natural coral, genus Porites. It is biocompatible and resorbable with a pore size of 100 to 200 □m, similar to the porosity of spongy bone (Guillemin G, Patat J.L., Fournie J., et al, 1987). In contrast to porous HA, derived from the same coral by heat conversion and nonresorable, calcium carbonate is resorbable.

HTR (Bioplant, Norwalk, CT) is a biocompatible microporous composite of polymethylmethacrylate (PMMA), polyhydroxylethylmethacrylate (PHEMA), and calcium hydroxide. Histologically, new bone growth has been found deposited on HTR particles (Guillemin G, Patat J.L., Fournie J., et al, 1987; Yukna R.A., Greer R.O. Jr, 1992).

Bioceramics alloplasts are primarily composed of calcium phosphate. The two most widely used forms are HA and tricalcium phosphate. Tricalcium phosphate is a porous form of calcium phosphate that is also referred to as □-calcium phosphate. The proportion of calcium and phosphate is similar to a bone. It serves as a biologic filler, which is partially resorbable and allows bone replacement of the implant material.

Generally, hydroxyapatite as a bone substitute is not osteoinductive but rather osteoconductive. When osteogenic

activity was observed, it usually has been reported with porous HA and not with dense HA.

There are two forms of bioactive glass currently available. PerioGlass(BioGlass synthetic bone graft particulate) and Biogran (resorbable synthetic bone graft). Bioactive glasses are composed of SiO2, CaO, Na2O, P2O5 and bond to bone through the development of a surface layer of carbonated HA (Hench L.L., Paschall H.A., 1973)

Guided Tissue Regeneration (GTR).

GTR involves the use of a barrier membrane to seal off a defect site during healing. This barrier, which is sutured in a tension-free fashion between the defect and thick reflected flaps, deters undesirable tissues that have no osteogenic potential, such as epithelium and soft connective tissue, from invading the wound site during healing. This, in turn, allows space for periodontal ligament cells to grow unimpeded and to form a new attachment apparatus.

GTR has been widely used to treat a variety of periodontal defects successfully (Gottlow J., Nyman S., Karring T., 1992). Three-walled defects respond best to GTR treatment, typically experiencing substantial bone fill (American Academy of Periodontology, Committee on Research, Science and Therapy: Periodontal Regeneration, Chicago, American Academy of Periodontology, 1993). Other defects that have been shown to respond well include combination two-walled and three-walled defects, funnel-shaped defects with definite osseous stops, and class II furcations with vertical components. When treating maxillary molars with class II defects, however, only buccal sites have shown positive responses (Garrett S., 1996). Any defects treated by GTR should be at least 5 mm deep (Becker W., 1994). First described by Nyman et al in 1982, GTR procedures initially employed nonbiodegradable

(nonabsorbable) barriers, that were surgically removed from the wound site after a period of healing. More recently, a number of absorbable barrier materials have been introduced that appear to offer certain advantages over traditional nonabsorbable materials (Gottlow J., 1993; Polson A.M., Garrett S., Stoller N.H., et al, 1995; Wang H.L., O'Neal R.B., Thomas C>L., et al, 1994).

Barrier techniques, using materials such as expanded polytetrafluoroethylene (ePTFE), polyglactin, polylactic acid, and collagen, are employed to exclude dental tissues with little or no regenerative capacity (i.e., gingival epithelium and corium) while permitting or encouraging wound repopulation by cells derived from tissues with known or suspected regenerative potential (PDL and alveolar bone). This approach is based on the concept that the type of periodontal tissue that forms on instrumented root surfaces after surgical treatment is determined by the type and origin of cells that migrate and attach to the root during early wound healing (Melcher A.H., 1976).

Collagen-based barriers belong to the absorbable barrier materials. The collagen found in GTR barriers can be of various subtypes (usually type I collagen is predominant) and can be derived from various animal sources. Collagen barriers are made by using extrusion-coagulation and air drying to form sheets of material from dilute (<1%) collagen solutions. Most barriers are cross-linked to extend the absorption time and to reduce antigenicity. Cross-linking can be controlled by physical (e.g., gamma or ultraviolet irradiation) or chemical (e.g., formaldehyde) processing methods.

Degradable polymers constitute the other major group of absorbable barrier materials used in clinical practice today. These polymer barriers are synthesized by copolymerization of different forms of polylactic acid (PLA), polyglycolic acid (PGA), or mixtures of PLA and PGA. Barrier degradation

occurs by hydrolysis of ester bonds, a process that requires 30-60-plus days depending on the polymeric composition of the material.

GTR barriers in future can serve as deliver agents and factors (e.g., antibiotics, growth factors, chemotactic factors, or adhesion factors) to the wound to orchestrate and direct natural wound healing better. These materials will then have to act not only as barriers, but also as delivery devices to release specific agents on a time or need basis (Wang H.L., MacNeil R.L., 1998).

2.4. Occlusal Adjustment

Prosthetic treatment is one of the necessary stages in complex treatment of periodontal diseases.

Indication for the open curettage is the II stage of periodontitis (periodontal pocket up to 5 mm) without infrabony pockets.

Prosthetic measures in periodontology help to renew occlusal relations and to stop teeth mobility by different ways of their stabilization. Prosthetic measures are mostly used in the case of secondary traumatic occlusion. Prosthetic appliances can be temporary and permanent. Temporary ones being used during therapeutic and surgical treatment and after the surgical phase in 2-3 weeks permanent prosthetic appliances should be fixed.

Orthodontic measures are often recommended in patients with malocclusion and healthy periodontal tissues, that is in the case of the primary traumatic occlusion. Nowadays, orthodontics is also widely used for occlusal adjustments in the periodontal pathology.

Occlusal adjustment is the establishment of functional relationships favourable to the periodontium by one or more of

the following procedures: reshaping of the teeth by grinding, dental restoration, tooth movement, tooth removal, or orthognathic surgery. ***Coronoplasty*** is the selective reduction of occlusal areas with the primary impose of influencing the mechanical contact conditions and the neural pattern of sensory input. It is a direct and irreversible change of the occlusal scheme. There is a tendency to think of occlusal adjustment solely as eliminating injurious occlusal forces, but an equally important purpose is to provide the functional stimulation necessary for the preservation of periodontal health.

Therapeutic occlusion is an occlusal treatment model used in restoring or replacing teeth so that the adaptation required of the patient and the need for compensatory tissue change are minimized.

The optimal design goal of a therapeutic occlusion is the elimination of undesirable occlusal supracontacts and the creation of a stable mandibular position. This ensures that jaw closure occurs at one functional end point wherein the temporomandibular joints (TMJs) and teeth receive stresses with only a slight mesial component.

Occlusion is adjusted for patients who demonstrate periodontal changes manifested in the form of excessive tooth mobility, angular thickening of the periodontal ligament, true instances of angular (vertical) bone destruction, furcation involvement, and migration of teeth.

The absence of signs of trauma from occlusion determines that the patient's occlusal relationships merit perpetuation in periodontal restorative therapy. If the trauma is limited to a single tooth or a few teeth, localized coronoplasty of supracontacts may suffice, and no change in the mandibular position need be made. However, if there is a generalized trauma from occlusion, faulty maxillomandibular relationships may be involved in the production of the trauma. Normalization of these relationships may require major

changes in the mandibular occlusal position in accordance with the guidelines for therapeutic occlusion to minimize the repetitive and excessive occlusal forces. However, periodontal changes such as an elevated periodontal/gingival index or increased pocket depth have not been associated with occlusal factors or even tooth mobility.

Splinting – connection of several teeth into united block with the purpose of limitation of teeth mobility and redistribution of functional loading.

Two types of splinting can be distinguished: temporary and permanent.

Both types of splinting should correspond to the certain demands:

1) to create stable block from the group of teeth, restricting their movements in three perpendicular directions,

2) to be rigid and fixed firmly on the teeth,

3) not to irritate marginal periodontium,

4) not to prevent from the possibility of therapeutic and surgical manipulations,

5) not to have retention points for food retardation,

6) not to increase the height of the lower third of the face and not to have premature occlusal contacts on its surface,

7) do not change the speech of patient,

8) teeth preparation have to be as minimum as possible.

Splints can be fixed or in the form of partial denture

Demands to the splints	Types of splints	
	In the form of partial dentures	Fixed splints
1. Restricting of teeth movements in three	Restrict teeth mobility in	Restrict teeth mobility

dimensions	horizontal surface	in three surfaces
2. Traumatization of marginal periodontium	Do not cause	Can cause
3. Prevention for the therapeutic and surgical treatment	Do not break	Can break
4. Hygiene of the oral cavity	Do not influence	Can influence
5. Necessity of teeth preparation	Absent or minimal preparation	Often into the large extend

Splint in the form of partial dentures are indicated:

1) in the generalized periodontitis with the even bone loss not more than 1/2 of the teeth roots,

2) in the initial stages of disease to remove or lower horizontal teeth overloading,

3) as prophylactic measures,

4) when partial dentures are indicated.

Fixed splints are recommended:

1) in uneven bone resorption more than 0,5 of teeth roots length,

2) localized process,

3) presence of teeth with different stages of mobility,

4) to remove overloading in vertical dimension.

Connectional effect of partial dentures is provided by the system of supporting retentional clasps and occlusal onlays united in the splint. This splint is made by casting.

Fixed splints for temporary use are made from composite material connected with the help of special tissues „Ribbond", „Connect", „GlassSpan".

Tooth tissues for splinting are prepared mostly on the palatal, lingual occlusal surface. The depth of preparation depends on the size of splinting device. After the preparation

teeth surfaces are etched and thoroughly rinsed by water, dried. These stages are followed by bonding procedure.

Prepared special wipe is covered by the adhesive agent and polymerized. Fixing agent then are put in prepared cavities filled with a thin layer of composite and polymerized. The rest cavities are filled with the composite and polymerized. Occlusal adjustment and polishing of the splint finish teeth connecting procedure.

2.5. Peculiarities in the Treatment of Periodontitis in I-st, II-nd and II-rd Stages of Heaviness

Treatment of the Generalized Periodontitis in the I-st stage of heaviness

Therapeutical phase: professional hygiene with detailed removal of subgingival calculus, the main local etiological factor of gingival inflammation; local antiinflammatory treatment, proteolytic enzymes locally when exudate is present in the periodontal pockets.

Surgical treatment: closed curretage.

General treatment: vitamins (adequate nutrition), treatment of general diseases.

Sometimes physical therapy is recommended to improve the treatment results, 10-14 days after periodontal surgery.

Electrophoresis and phonophoresis are used to enrich periodontal tissues with vitamins, minerals, microelements more effectively than by the way of gingival applications.

Treatment of the Generalized Periodontitis in the II-nd stage of heaviness

In therapeutic phase of treatment of this stage of periodontitis, except detailed calculus removal, antimicrobial

remedies and antibiotics are prescribed, especially in the cases of purulent pockets exudate present and abscess formation. Antibiotics with the particular deposition in the bone (lincomycini, tetracyclini, metacyclini, oletetrini, rondomicini) are used and among the antimicrobial remedies - metronidazolum is effective against pathogenic pocket microbiota. When used locally lincomycin is prescribed as submucosal injections (300 mg on each quadrant with 0,5 ml of anesthetic). Injections are made in two-three days one time in one teeth quadrant.

Antiinflammatory nonsteroid medications are used to diminish the inflammation in periodontal tissues. Locally they are used in periodontal dressings.

Physical therapy: electrophoresis with heparin to improve microcirculation in periodontal tissues, ambeni (depresses fibrinolysis processes) are sometimes recommended. After surgical treatment electrophoresis with 10% Ca gluconatis and 1% NaF can be recommended. Biogenic stimulators (aloe, gumizol and others) can be also given in the form of electrophoresis.

In prosthetic treatment the temporary and permanent splinting is very important, because of pronounced teeth mobility in this stage of periodontitis. Temporary splinting being done usually before a surgical phase of treatment, sometimes even before the therapeutic phase. Permanent splinting and bridges are prepared in one-two months after surgical treatment.

Surgical phase in the II stage of periodontitis includes open curretage (in the regions with prominent gingival recession) and periodontal flaps. During surgery osteoplastic procedures are recommended in deep infrabone pockets.

General therapy is very important in the II stage of periodontitis. Periodontitis has often to consult specialists in gastroenterology, endocrinology, immunology.

Balanced nutrition, vitamin therapy (preferably in the form of injections) is recommended.

Treatment of the Generalized Periodontitis in the III-rd stage of heaviness

Therapeutical stage: preparing of the patient for periodontal surgery. Antimicrobial therapy (local and general), antiinflammatory therapy, professional hygiene. Prosthetic treatment before the surgery - temporary splinting.

Surgical treatment: flap operations with stimulation of bone regeneration (autotransplantation, allotransplantation, biomembranes,etc.).

After the periodontal surgery antimicrobial, antiinflammatory treatment is continued.

Prosthetic treatment includes permanent splinting, bridges one-two months after surgery, prosthodontic treatment 3-5 months after surgery.

General treatment in gastroenterologist, endocrinologist, balanced nutrition, etc. is conducted constantly during the whole period of periodontal treatment.

Questions

1. What does it mean that treatment of the periodontitis has to be complex?
2. What types of surgery procedures are used in periodontology?
3. Describe step-by-step closed curettage technique.
4. What are the indications to open curettage?
5. Describe the procedure of modified Widman flap.
6. What is the plan of treatment in the Generalized Periodontitis of the I-st stage of heaviness.

7. What is the plan of treatment in the Generalized Periodontitis of the II-nd stage of heaviness.
8. What is the plan of treatment in the Generalized Periodontitis of the III-rd stage of heaviness.

2.6. Physiotherapy in the Periodontal Diseases

Procedures based on the influence of constant current.

Halvanization (constant current of 50 mA); improve the metabolic processes in the tissues, normalize blood and lymphocirculation. This method is used in the diseases with the trophic changes in the tissue - as periodontitis and periodontosis, mostly in the stage of maintenance care.

Electrophoresis – method of the introduction of some medications into the tissues with the help of constant current. The method of electrophoresis is based on the peculiarities of the substances to dissociate into ions.

Medications can be also introduced into the tissues with the help of ultrasound - ultraphonophoresis. For example ultraphonophoresis of colanchoe (5 ml of siccus colanchoe, 5 ml of distilled water and 5 g of lanolini), ultraphonophoresis of 5% ascorbinic acid (5% solution of ascorbinic acid with 20 ml of oleum persicorum).

Intensity of ultrasound vibrations are 0,2-0,4 watt/cm^2.

Ultraphonophoresis of lidase is used to dissolve scar tissues.

Ultraphonophoresis of herapini ointment, of vitamins A, E, K and other medication are the alternatives to the electrophoresis.

Diadynamotherapy – method of electrotherapy with low current intensity and voltage, based on the influence of polysynusoidal current with low frequency. Diadynamic

currents have positive influence on the blood circulation and metabolic processes, are effective in pain reduction.

Darsonvalinization – method of electrotherapy with the influence of high frequency and high voltage alternative current. Current influences the tissues with impulses and has the effect of electromassage, stimulating effect, leading to vessels broadening, and intensification of metabolism in periodontal tissues . Method is especially recommended in periodontosis.

Ultra high frequency therapy and superhigh frequency therapy has prominent antiinflammatory effects and induce infiltrate dissolvation, reduce pain. High frequency influences lead to the decrease tissue permeability and reduction of inflammatory mediators in the tissues. It has a slight heating effect.

Light treatment

Infrared radiation

Infrared light penetrates the tissues 3-4 mm in depth. Being absorbed by the tissues, infrared radiation is transformed to heat energy. This effect is used in treatment of chronic and subacute inflammatory processes, in the case of infiltrates present after operations, in arthritis, neuritis and myositis pain.

Ultraviolet radiation

This type of radiation has bactericidal effect and increases the reactiveness of the body, normalizes metabolic processes, erythropoesis, lower the serum cholesterol level. This therapy is used in multicaries lesions, chronic recurrence aphthous stomatitis, multiform exudative erythaema, herpetic lesions. It is very important to estimate the individual biodose. Irradiation is recommended to be started with 1/2 of individual biodose and finished in several visits in 3-4 biodoses.

2.7. Maintananace Care in the Periodontal Teatment

All the patients with periodontal diseases are dispensarizised, that means they have to come for compulsory examinational check-ups. All patients can be distributed in three main groups (Danylevskyj).

Group one – healthy persons, without periodontal diseases - control visits once a year.

Group two – patients with the high risk of periodontal diseases, local risk factors - maloocclusions and general factors - somatic pathology and patients treated for periodontal diseases after the achievement of the process stabilization for one year in gingivitis and two years in periodontitis.

Group three – patients treated for periodontal diseases.

Subgroup A – patients with active processes being treated at present

Subgroup B – patients which have finished the complex treatment of the disease achieving disease remission. The term remission means clinical improvement (absence of bleeding, periodontal pockets, normal gingival tissues but often with the gingival recession present after the treatment) and normalization of the bone structure. Patients in subgroup B have to come for examinational check-ups once a year in gingivitis, twice a year in periodontitis stage II and 3-4 times a year in periodontitis stage III. After the achieving of stable remission for one year in gingivitis and two years in periodontitis patients can be transformed from the group III B to the group II.

After the complex therapy in patients with generalized periodontitis has been carried out, the question, concerning the maintenance therapy with the account of the level of heaviness and character of the disease, arises.

There is a great necessity to either eliminate the cause of the recurrence of disease or to minimize the influence of it.

Two moments are to be precisely distinguished, thereat: the relapse of the inflammatory process in the periodontal tissues and the relapse of the periodontitis, that is the progressing of the periodontal, inflammatory-destructive process with the additional loss of the attachment and the formation (or deepening of the previously existing) periodontal pocket.

As a rule, in the first clinical situation, it is quite enough to use the local measures (the tooth surface debridment, the polishing of the roots, the antiinflammatory therapy), while the second one demands the extra use of the surgical methods including the osteotrophic and stimulating therapy. The periodontal practice points out the five main causes leading to the renewal of the activity of the generalized periodontitis.

They are as follows: the reduction of the non-specific resistence of the organism; the exacerbation of the somatic pathology with the aggrevation of the secondary immunodeficiency that accompanies it; the long-term stress reactions; denaturalization of the nutrition as well as the poor oral hygiene.

Therefore, taking into account the above-mentioned reasons, the use of the antibacterial, immunomodulatory, antioxidative or health-restorative therapy is strongly recommended along with the rotational diet and means for the deliverance of the internal medium acidosis.

3. PERIODOTOSIS

3.1. The Main Clinical Signs

Periodontosis is considered to be a generalized dystrophic process in periodontal tissues.

Etiology of periodontosis is associated with neurotrophic changes (Danilevskyj's, theory) and changes in blood vessels of periodontium leading to reduced vascularization and thus to dystrophic changes in the tissues (Evdokimov's, theory).

Gums are often pale and thin (atrophic gingivitis), bleeding is not a typical sign. Gingival recession without pocket formation, horizontal bone loss and osteosclerosis, traumatic occlusion are always present in the periodontosis patients. When compared with periodontitis, the teeth mobility is less in periodontosis, and occurs only in late stages of the disease. Among the changes in tooth hard tissues pathological attrition, wedge-shaped non-caries defects, and denticles in pulp chamber are typical for periodontosis. Hypercementosis, seen in the x-ray as the narrowing of periodontal slit is one of the typical sign in periodontosis.

Clinically, heaviness of the periodontosis depends on the level of bone resorption and gums recession.

In the I-st stage of disease bone destruction is 1/3 of tooth root, only teeth necks are denuded and patient sometimes complains of the teeth hypersensitiveness.

The II-nd stage of disease is characterized by teeth sensitivity, bone destruction - up to 1/2 of the teeth roots, pathological attrition of the teeth, gums recession 3-4 mm (fig.42, 43).

In the III-rd stage of periodontitis, the bone loss is more than 1/2 of the teeth roots, severe recession of the gums is connected with teeth attrition. At this stage, there is a tendency to the mobility of teeth.

Treatment of the periodontosis is symptomatic to a large extent and includes the remineralization of teeth and filling of the wedge-shaped defects. The treatment can also provide the procedures, stimulating metabolic processes in periodontal tissues: electrophoresis with biostimulators (phibs,

aloe), massages of the gums: automassage, hydromassage, electromassage (darsonvalization), vacuummassage, electrophoresis with Ca glycerophosphatis and NaF. In general, in the treatment of periodontosis it is very important to improve metabolic processes in the body and to control the serum cholesterol level as one of the reason of atherosclerosis.

Questions:

1. Characterise the changes taking place in the periodontal tissues during the Periodontosis?
2. What are the main principles of treatment of the Periodontosis?

4. PROGRESSING IDIOPATHIC DISEASES OF THE PERIODONTIUM

4.1. The Peridontium in Hematologic Diseases

Leukemia

The leukemias are "malignant neoplasias of white blood cell precursors, characterized by (1) diffuse replacement of the bone marrow with proliferating leukemic cells; (2) abnormal numbers and forms of immature white cells in the circulating blood; and (3) widespread infiltrates in the liver, spleen, lymph nodes and other sites throughout the body.

According to the type of white blood cell involved, leukemias can be ***lymphocytic*** or ***myelocytic***; a subgroup of the myelocytic leukemias is ***monocytic leukemia***. According to their evolution, leukemias can be ***acute***, which is rapidly fatal; ***subacute***; or ***chronic***. The replacement of the bone marrow

elements by leukemic cells reduces normal white blood cell and platelet production, leading to anaemia and bleeding disorders. Some patients may have normal blood counts while leukemic cells are present in the bone marrow; this type of disease is called ***aleukemic leukemia***.

Oral and periodontal manifestations of leukemia consist of the following: leukemic infiltration of the periodontium, bleeding, oral ulcerations and infections. Leukemic cells can infiltrate the gingiva and, less frequently, the alveolar bone. Gingival infiltration often results in ***leukemic gingival enlargement***.

Leukemic gingival enlargement is not found in edentulous patients or in patients with chronic leukemia. Leukemic gingival enlargement consists of a basic infiltration of the gingival corium by leukemic cells that creates gingival pockets where bacterial plaque accumulates, initiating a secondary inflammatory lesion that contributes also to the enlargement of the gingiva.

Clinically, the gingiva appears initially bluish red and cyanotic, with a rounding and tenderness of the gingival margin; then it increases in size, most often in the interdental papilla and partially covering the crowns of the teeth.

Microscopically, the gingiva exhibits a dense, diffuse infiltration of predominantly immature leukocytes in the attached as well as the marginal gingiva. Occasional mitotic figures indicative of ectopic hematopoiesis may be seen. The normal connective tissue components of the gingiva are displaced by the leukemic cells. The nature of the cells depends on the type of leukemia. The cellular accumulation is denser in all the reticular connective tissue layer. In almost all cases, the papillary layer contains comparatively few leukocytes. The blood vessels are distended and contain predominantly leukemic cells, and the red blood cells are reduced in number. The epithelium presents a variety of changes. It may be thinned

or hyperplastic. Degeneration associated with intercellular and intracellular oedema and leukocytic infiltration with diminished surface keratinization are common findings.

The microscopic picture of the marginal gingiva differs from that of the remainder of the gingiva in that it usually exhibits a notable inflammatory component in addition to the leukemic cells. Scattered foci of plasma cells and lymphocytes with oedema and degeneration are common findings. The inner aspect of the marginal gingiva is usually ulcerated, and marginal necrosis with pseudomembrane formation may also be seen.

The periodontal ligament and alveolar bone may also be involved in acute and subacute leukemia. The periodontal ligament may be infiltrated with mature and immature leukocytes. The marrow of the alveolar bone exhibits a variety of changes, such as localized areas of necrosis, thrombosis of the blood vessels, infiltration with mature and immature leukocytes, occasional red blood cells, and replacement of the fatty marrow by fibrous tissue.

In leukemic mice, the presence of infiltrate in marrow spaces and the periodontal ligament results in osteoporosis of the alveolar bone with destruction of the supporting bone and disappearance of the periodontal fibers.

The abnormal accumulation of leukemic cells in the dermal and subcutaneous connective tissue is called ***leukemia cutis*** and forms elevated flat macules and papules.

Bleeding. Gingival hemorrhage is a common finding in leukemic patients, even in the absence of clinically detectable gingivitis. Bleeding gingiva can be an early sign of leukemia. It is due to the thrombocytopenia that results from replacement of the bone marrow cells by leukemic cells and also from the inhibition of normal stem cell function by leukemic cells or their products.

Oral Ulcerations and Infections. The granulocytopenia resulting from the replacement of bone marrow cells by leukemic cells reduces the tissue resistance to opportunistic microorganisms and leads to ulcerations and infections. Discrete, punched-out ulcers penetrating deeply into the submucosa and covered by a firmly attached white slough can be found in the oral mucosa. These lesions occur in sites of trauma such as the buccal mucosa in relation to the line of occlusion or the palate. Patients with past history of herpes infection may develop herpetic oral ulcers, frequently in multiple sites and large atypical forms, after chemotherapy is instituted.

Gingival bacterial infection in leukemic patients can be a primary bacterial infection or result from an increased severity of existing gingival or periodontal disease. Lesions of acute necrotizing ulcerative gingivitis may also be seen in terminal cases of leukemia.

In leukemia, the response to irritation is altered, so that the cellular component of the inflammatory exudate differs both quantitatively and qualitatively from that that occurs in nonleukemic individuals. There is a pronounced infiltration of immature leukemic cells in addition to the usual inflammatory cells.

The inflamed gingiva differs clinically from inflamed gingiva in nonleukemic individuals. It is a peculiar bluish red, is markedly sponge-like and friable, and bleeds persistently on the slightest provocation or even spontaneously. This markedly altered and degenerated tissue is extremely susceptible to bacterial infection, which can be so severe as to cause acute gingival necrosis and pseudomembrane formation. These are secondary oral changes superimposed on the oral tissues altered by the blood disturbance and produce associated disturbances that may be a source of considerable difficulty to the patient, such as systemic toxic effects, loss of appetite,

nausea, blood loss from persistent gingival bleeding, and constant gnawing pain. By eliminating local irritants, it is possible to alleviate severe oral changes in leukemia.

Chronic Leukemia

In chronic leukemia, clinical oral changes suggesting a hematologic disturbance are very rare.

The microscopic changes in chronic leukemia may consist of replacement of the normal fatty marrow of the jaws by islands of mature lymphocytes or lymphocytic infiltration of the marginal gingiva without dramatic clinical manifestations.

Gingival Biopsy and Leukemia

The existence of leukemia is sometimes revealed by a gingival biopsy performed to clarify the nature of a troublesome gingival condition. In such cases, the gingival findings must be corroborated by medical examination and hematologic study. The absence of leukemic involvement in a gingival biopsy specimen does not rule out the possibility of leukemia. In chronic leukemia, the gingiva may simply present inflammatory changes, with no suggestion of a hematologic disturbance. In patients with recognized leukemia, the gingival biopsy indicates the extent to which leukemic infiltration is responsible for the altered clinical appearance of the gingiva. *Although such findings are of interest, their benefit to the patient is insufficient to warrant routine gingival biopsy studies in patients with leukemia.*

Anaemias

Anaemias are deficiencies in the quantity or quality of the blood as manifested by a reduction in the number of erythrocytes and in the amount of hemoglobin. Anaemia may be the result of blood loss, defective blood formation, or increased blood destruction.

Anaemias are classified according to cellular morphology and hemoglobin content as (1) macrocytic hyperchromic anaemia (pernicious anaemia), (2) microcytic hypochromic anaemia (iron deficiency anaemia), (3) sickle cell anaemia, or (4) normocytic-normochromic anaemia (hemolytic or aplas-tic anaemia).

Pernicious anaemia results in tongue changes in 75% of cases. The tongue appears red, smooth, and shiny owing to atrophy of the papillae. There is also marked pallor of the gingiva.

Iron deficiency anaemia induces similar tongue and gingival changes. A syndrome consisting of glossitis and ulceration of the oral mucosa and oropharynx, inducing dysphagia (***Plummer-Vinson syndrome***), has been described in patients with iron deficiency anaemia.

Sickle cell anaemia is a hereditary form of chronic hemolytic anaemia that occurs almost exclusively in blacks. It is characterized by pallor, jaundice, weakness, rheumatoid manifestations, and leg ulcers. Oral changes include generalized osteoporosis of the jaws, with a peculiar stepladder alignment of the trabeculae of the interdental septa and pallor and yellowish discoloration of the oral mucosa. Periodontal infections may precipitate sickle cell crisis. ***Aplastic anaemias*** result from a failure of the bone marrow to produce erythrocytes. Their etiology is usually the effect of toxic drugs on the marrow. Oral changes include pale discoloration of the oral mucosa and increased susceptibility to infection owing to the concomitant neutropenia.

Thrombocytopenic Purpura

Thrombocytopenic purpura may be idiopathic (i.e., of unknown etiology, as in Werlhof's disease), or it may occur secondary to some known etiologic factor responsible for a reduction in the amount of functioning marrow and a resultant

reduction in the number of circulating platelets. Such etiologic factors include aplasia of the marrow; crowding out of the megakaryocytes in the marrow, as, for example, in leukemia; replacement of the marrow by tumor; and destruction of the marrow by irradiation or radium or by drugs such as benzene, aminopyrine, and arsenical agents.

Thrombocytopenic purpura is characterized by a low platelet count, a prolonged clot retraction and bleeding time, and a normal or slightly prolonged clotting time. There is spontaneous bleeding into the skin or from mucous membranes. Petechiae and hemorrhagic vesicles occur in the oral cavity, particularly in the palate and the buccal mucosa. *The gingivae are swollen, soft, and friable. Bleeding occurs spontaneously or on the slightest provocation and is difficult to control.* Gingival changes represent an abnormal response to ***local irritation***; the severity of the gingival condition is dramatically alleviated by removal of the local irritants.

4.2. The Periodontium in the Immunodeficiency Diosordes

Deficiencies in host defense mechanisms may lead to severely destructive periodontal lesions. These deficiencies may be primary, or inherited; or secondary, caused by immunosuppressive drug therapy or pathologic destruction of the lymphoid system. Leukemia, Hodgkin's disease, lymphomas, and multiple myeloma all may result in secondary immunodeficiency disorders.

Leukocyte disorders. Disorders that affect production or function of leukocytes may result in severe periodontal destruction.

Agranulocytosis. Agranulocytosis is characterized by a reduction in the number of circulating granulocytes and results

in severe infections, including ulcerative necrotizing lesions of the oral mucosa, skin, and gastrointestinal and genitourinary tracts. Less severe forms of the disease are called neutropenia or granulocytopenia.

Drug idiosyncrasy is the most common cause of agranulocytosis, but in some instances its etiology cannot be explained. Agranulocytosis has been reported after the administration of drugs such as aminopyrine, barbiturates and their derivatives, benzene ring derivatives, sulfonamides, gold salts, or arsenical agents. It generally occurs as an acute disease, but it sometimes reappears in cyclic episodes (***cyclic neutropenia***). It may be periodic with recurring neutropenic cycles.

The onset of the disease is accompanied by fever, malaise, general weakness, and sore throat. Ulceration in the oral cavity, oropharynx, and throat is characteristic. The mucosa exhibits isolated necrotic patches that are black and grey and are sharply demarcated from the adjacent uninvolved areas. The absence of a notable inflammatory reaction because of lack of granulocytes is a striking feature. The gingival margin may or may not be involved. Gingival hemorrhage, necrosis, increased salivation, and fetid odour are accompanying clinical features. The occurrence of rapidly destructive periodontitis has been described in cyclic neutropenia.

Because infection is a common feature of agranulocytosis, differential diagnosis involves consideration of such conditions as acute necrotizing ulcerative gingivitis, diphtheria, noma, and acute necrotizing inflammation of the tonsils. Definitive diagnosis depends on the hematologic findings of pronounced leukopenia and almost complete absence of neutrophils.

Human Immunodeficiency Virus

The oral manifestations of HIV infection are mainly the result of cellular immunodeficiency induced by HIV and may be seen with variable frequency. Oral manifestations of HIV infection can be divided into three categories. Group 1 lesions are strongly associated with HIV infection, and group 2 lesions are less commonly associated with HIV infection, and group 3 lesions are seen in HIV infection.

In oral cavity candidiasis (erythematous, pseudomembranous) is the most common feature in group 1. Oral candidiasis is usually associated with the progression of HIV disease and could also be considered a predictor for development of acquired immunodeficiency (AIDS).

Hairy leukoplakia, Kaposi's sarcoma, Non-Hodgkin's lymphoma are common in high-risk group for HIV infection.

Among bacterial infections, periodontal disease is relatively common in HIV-infected individuals. Periodontal diseases in group 1 lesions are: linear gingival erythema, necrotizing (ulcerative) gingivitis, necrotizing (ulcerative) periodontitis.

Clinically, linear gingival erythema is characterized by a fiery red band along the margin of the gingival. The lesion does not respond to plaque control measures or root planing and scaling. Gingival bleeding may occur spontaneously or on probing. Necrotizing periodontitis is characterized by soft ulceration and necrosis and rapid destruction of the periodontal attachment apparatus. Spontaneous bleeding and severe deep pain are common. The condition does not respond to conventional treatment. Necrotizing periodontitis is usually localized although in severe cases it may be generalized. The possibility of HIV infection must be considered, particularly if high-risk behavior is associated with necrotizing gingivitis. Necrotizing gingivitis may in some cases progress to necrotizing stomatitis. The latter is characterized by localized

acute ulceronecrotic lesions of the oral mucosa. The underlying bone is exposed or penetrated.

In group 2 (lesions less commonly associated with HIV infection) necrotizing (ulcerative) stomatitis, melanotic hyperpigmentation, dry mouth due decreased salivary flow rate, unilateral or bilateral swelling of major salivary glands, thrombocytopenic purpura, bacterial infections (mycobacterium avium-intracellulare, mycobacterium tuberculosis), viral infections (herpes simplex virus, human pappiloma virus, varicella-zoster virus) can be revealed.

Oral infections with actinomyces israelii, escherichia coli, klebsiella pneumoniae have rarely been reported in HIV-infected patients (group 3 – lesions seen in HIV infection). Drug reactions, fungal infection other than candidiasis, neurologic disturbances (facial palsy, trigeminal neuralgia), viral infections (cytomegalovirus, molluscum contagiosum) may represent manifestations of HIV infections.

4.3. Diabets and the Periodontium

Diabetes is an extremely important disease from a periodontal standpoint. It is a complicated metabolic disease characterized by hypofunction or lack of function of the cells of the islets of Langerhans in the pancreas, leading to high blood glucose levels and excretion of sugar in the urine. Two basic types of primary diabetes mellitus have been described: insulin-dependent and non-insulin-dependent.

Insulin-dependent diabetes mellitus (IDDM) (type I) is also known as juvenile diabetes or juvenile-onset diabetes, although it may sometimes appear at older ages. This type of diabetes results from an absolute lack of insulin, is very unstable and difficult to control, has a marked tendency toward ketosis and coma, is not preceded by obesity, and requires

injected insulin to be controlled. Patients with the disease present with the symptoms traditionally associated with diabetes: polyphagia, polydipsia, polyuria, predisposition to infections, and anorexia.

Non-insulin-dependent diabetes mellitus (NIDDM) (type II) is the adult type (i.e., onset usually after age 45). It generally occurs in obese individuals and can often be controlled by diet or by oral hypoglycemic agents. The development of ketosis and coma is not common. Adult-onset diabetes has the same symptoms as juvenile diabetes but in a less severe form.

Oral Manifestations of Diabetes

The following findings have been described in the oral mucosa: cheilosis and a tendency toward drying and cracking; burning sensations; decrease in salivary flow; and alterations in the flora of the oral cavity, with greater predominance of Candida albicans, hemolytic streptococci, and staphylococci. These changes, however, are not specific, and terms such as diabetic stomatitis should not be used. Perhaps the most striking changes in uncontrolled diabetes are the reduction in defense mechanisms and the increased susceptibility to infections leading to destructive periodontal disease. Control of diabetes may be attained by diet or by the administration of insulin and/or other drugs. In well-controlled diabetes, none of the previously mentioned changes is found. There is a normal tissue response, no increase in the incidence of caries, a normally developed dentition, and a normal defense against infections. However, the possibility that the control of the disease may be inadequate makes it advisable to exercise special care in the periodontal treatment of individuals with controlled diabetes.

A variety of periodontal changes have been described in diabetic patients, such as a tendency toward abscess formation, diabetic periodontoclasia, enlarged gingiva, sessile or

pedunculated gingival polyps, polypoid gingival proliferations, and loosened teeth. Very severe gingival inflammation, deep periodontal pockets, rapid bone loss, and frequent periodontal abscesses often occur in diabetic patients with poor oral hygiene. The distribution and severity of local irritants affect the severity of periodontal disease in diabetics. Diabetes does not cause gingivitis or periodontal pockets, but there are indications that it alters the response of the periodontal tissues to local irritants, hastening bone loss and retarding postsurgical healing of the periodontal tissues. Frequent periodontal abscesses appear to be an important feature of periodontal disease in diabetics.

4.4. Papillon-Lefevre Syndrome

This autosomal recessive disorder predominantly demonstrates oral and dermatologic manifestations. Because of the autosomal recessive inheritance pattern, the parents are not typically affected. The predominant oral finding is accelerated periodontitis that appears to be caused by defects in neutrophil function and multiple immune mediated mechanisms.

In most cases, the dermatologic manifestations become clinically evident in the first 3 years of life. Diffuse transgredient (first occurs on the palms and soles and then spreads to the dorsa of the hands and feet) palmar-plantar keratosis develops, with occasional reports of diffuse follicular hyperkeratosis and keratosis on the elbows and knees. Other less common sites of involvement include the legs, thighs, dorsal surface of the fingers and toes, and (rarely) the trunk. Although the appearance of the dermatologic manifestations is variable, the lesions typically present as white, light yellow, brown, or red plaques and patches that develop crusts, cracks, or deep fissures. Some patients describe worsening in the

winter, and others describe keratotic desquamation, which may be confused with psoriasis.

The oral manifestations consist of dramatically advanced periodontitis that is seen in both the deciduous and permanent dentitions and develops soon after the eruption of the teeth. Extensive hyperplastic and hemorrhagic gingivitis is seen. A rapid loss of attachment occurs, with the teeth soon lacking osseous support and radiographically appearing to float in the soft tissue. Without aggressive therapy, the loss of the dentition is inevitable. Mobility and migration of the teeth are observed consistently, and mastication often is painful because of the lack of support. The teeth spontaneously exfoliate or are removed because of sensitivity during function. This process prematurely eliminates the deciduous dentition; with eruption of the permanent teeth, the destructive pattern is duplicated. When the teeth are absent, the alveolar mucosa is normal in appearance.

Although other pathogenic bacteria have been isolated from sites of active disease, A. actinomycetemcomitans has been related directly to the periodontal destruction. Although there is a hereditary component and leukocyte dysfunctions can be demonstrated, it appears that there must be an infection with a specific, potent bacterium, such as A. actinomycetemcomitans, for the periodontal component to develop.

The most successful treatment of the skin lesions has been administration of retinoids (e.g., etretinate), which has resulted in remarkable improvement with complete clearance in the majority of patients.

Attempts at resolution of the periodontal disease often have been frustrating. In spite of extensive periodontal therapy and antibiotics, in many patients the disease progresses until all teeth are lost. However, several investigators have reported a

cessation of attachment loss, and two different treatment approaches have been used.

Despite the use of numerous antibiotics, several reports document a difficulty in resolution of the infection associated with teeth that already exhibit attachment loss. In some of the cases, all of the periodontally involved deciduous teeth were extracted and followed by a period of edentulousness with antibiotic treatment in an attempt to remove the causative pathogens. Tetracycline was successful in preventing the redevelopment of periodontitis in the permanent teeth after the extractions and the resolution of the infection in the deciduous dentition. However, penicillin, erythromycin, metronidazole, and tetracycline were all unsuccessful in resolving active sites of periodontitis.

The second approach revolves around direct attack against A. actinomycetemcomitans. In one investigation, culture and sensitivity testing revealed that the most effective antibiotic regimen was amoxicillin and clavulanate potassium. Ceftriaxone and erythromycin were moderately effective; penicillin, tetracycline, and chloramphenicol were less effective. Metronidazole and ornidazole were ineffective. In another report, sensitivity testing suggested use of erythromycin and tetracycline; however, inadequate clearance of A. actinomycetemcomitans was seen after use of minocycline followed by erythromycin. Ultimately, clearance was achieved through mechanical removal combined with ofloxacin. It appears clear that clearance of A. actinomycetemcomitans is mandatory, but the antibiotic best suited for this task is variable.

Through the use of mechanical plaque control and appropriate antibiotics directed toward A. actinomycetemcomitans, the course of the disease may be altered. The progression of attachment loss is slowed dramatically, and the teeth that erupt after the initiation of

therapy do not develop periodontal destruction. Rigorous oral hygiene, chlorhexidine mouth rinses, frequent professional prophylaxis, and periodic appropriate antibiotic therapy are necessary for long-term maintenance.

4.5. The Lipid Reticuleoendoendothelioses

The Lipid Reticuloendothelioses are a relatively rare group of inherited disorders. They include the conditions known as the following: Gaucher disease, Niemann-Pick disease, Tay-Sachs disease.

Affected patients lack certain enzymes necessary for processing specific lipids, and this results in an accumulation of the lipids within a variety of cells. Because of this accumulation, it appeared that cells were attempting to store these substances; therefore, the term “storage disease” was commonly used for these disorders.

In ***Gaucher disease*** (the most common of the reticuloendothelioses), a lack of glucocerebrosidase results in the accumulation of glucosylceramide, particularly within the lysosomes of cells of the macrophage and monocyte lineage.

Niemann-Pick disease is characterized by a deficiency of acid sphingomyelinase, resulting in the accumulation of sphingomyelin, also within the lysosomes of macrophages.

Tay-Sachs disease is caused by a lack of hexosaminidase A, which results in the accumulation of a ganglioside, principally within the lysosomes of neurons.

All these disorders are inherited as autosomal recessive traits. When the genetic mutation known to cause Gaucher disease was evaluated for the Ashkenazi Jewish population, it was found that approximately 1 in 10 persons carried the defective gene. Most of the persons identified as having the

gene, however, were heterozygous and, therefore, asymptomatic.

The clinical features of Gaucher disease are generally caused by the effects of the abnormal storage of glucosylceramide. Macrophages laden with this glucocerebroside are typically rendered relatively nonfunctional, and they tend to accumulate within the bone marrow of the affected patient. This accumulation displaces the normal hematopoietic cells and produces anaemia and thrombocytopenia. In addition, these patients are susceptible to bone infarctions. The resulting bone pain is often the presenting complaint. Characteristic ***Erlenmeyer flask*** deformities of the long bones, particularly of the femur, are often identified. Accumulations of the macrophages in the spleen and liver result in visceral enlargement. Many affected patients show a significant degree of growth retardation. Neurologic deterioration may also occur in a few patients. Jaw lesions typically appear as ill-defined radiolucencies that usually affect the mandible without causing devitalization of the teeth or resorption of the lamina dura.

Niemann-Pick disease occurs as four different types, each associated with a different clinical expression and prognosis. Types A and B are caused by a deficiency of acid sphingomyelinase, whereas types C and D are due to mutations of NPC-1, a gene involved with cholesterol processing. Types A, C, and D have neuronopathic features, characterized by psychomotor retardation, dementia, spasticity, and hepatosplenomegaly, with death occurring during the first or second decade of life. Type B patients normally survive into adulthood and exhibit visceral signs, primarily hepatosplenomegaly, and sometimes pulmonary involvement.

Tay-Sachs disease may have a wide clinical range because the condition is genetically heterogeneous. Some forms are mild, with patients surviving into adulthood. In the

severe infantile form, however, rapidly progressive neuronal degeneration develops shortly after birth. Signs and symptoms include blindness, developmental retardation, and intractable seizures. Death usually occurs by 3 to 5 years of age.

Treatment and Prognosis of the Lipid Reticuloendothelioses

For patients with a mild expression of Gaucher disease, no treatment may be necessary. For more severe forms of Gaucher disease, enzyme replacement therapy with macrophage-targeted glucocerebrosidase is used. After 9 to 12 months of therapy, patients exhibit improvement in the status of their anaemia, a decrease in plasma glucocerebroside levels, and a decrease in hepatosplenomegaly. Resolution of the radiographic bone changes takes place over a longer period. Children treated with this regimen may show significant gain in height. Bone marrow transplantation has also been attempted; however, the problems inherent in graft-versus-host disease (GVHD) are still present with that form of therapy. A case-control study showed that adults with Gaucher disease have an increased risk for hematologic malignancies, particularly lymphoma and multiple myeloma. Genetic counseling should be provided to all affected patients.

The neuronopathic forms of Niemann-Pick disease and the infantile form of Tay-Sachs disease are associated with a poor prognosis. Genetic counseling should be provided for affected families. Molecular markers of these disorders have been developed to identify carriers. Such identification allows earlier intervention in terms of counseling, and targeted population screening for the gene that causes Tay-Sachs disease has resulted in a marked decrease in affected patients during the past 3 decades.

4.6. Histiocytosis X

The term histiocytosis X (***Langerhans Cell Histiocytosis; Langerhans Cell Disease; Idiopathic Histiocytosis, Eosinophilic Granuloma; Langerhans Cell Granuloma***) was introduced as a collective designation for a spectrum of clinicopathologic disorders characterized by proliferation of histiocyte-like cells that are accompanied by varying numbers of eosinophils, lymphocytes, plasma cells, and multinucleated giant cells. The distinctive histiocytic cells present in this lesion have been identified as Langerhans cells, and many believe that the condition is best designated as ***Langerhans cell histiocytosis***. Langerhans cells are dendritic mononuclear cells normally found in the epidermis, mucosa, lymph nodes, and bone marrow. These cells process and present antigens to T lymphocytes. For many years, it has been debated whether Langerhans cell histiocytosis represents a nonneoplastic condition or a true neoplasm. Studies examining the clonality of the lesional cells of this condition have shown this to be a monoclonal proliferation, a finding that is more consistent with a neoplastic process.

Clinical and Radiographic Features

The clinicopathologic spectrum traditionally considered under the designation of Langerhans cell histiocytosis includes the following:

– ***Monostotic or polyostotic eosinophilic granuloma of bone*** – solitary or multiple bone lesions without visceral involvement

– ***Chronic disseminated histiocytosis*** – a disease involving bone, skin, and viscera (Hand-Schüller-Christian disease)

– ***Acute disseminated histiocytosis*** – a disease with prominent cutaneous, visceral, and bone marrow involvement occurring mainly in infants (Letterer-Siwe disease)

It is difficult to categorize many patients into one of these classic designations because of overlapping clinical features. The often-cited Hand-Schüller-Christian triad – a bone lesions, exophthalmos, and diabetes insipidus – is present in only a few patients with chronic disseminated disease.

Although Langerhans cell histiocytosis may be encountered in patients over a wide age range, more than 50% of all cases are seen in patients under age 10. There is a definite male predilection. Bone lesions, either solitary or multiple, are the most common clinical presentation. Lesions may be found in almost any bone, but the skull, ribs, vertebrae, and mandible are among the most frequent sites. Children younger than age 10 most often have skull and femoral lesions; patients over age 20 more often have lesions in the ribs, shoulder girdle, and mandible. Adult patients with solitary or multiple bone lesions may have lymphadenopathy but usually do not have significant visceral involvement.

The jaws are affected in 10% to 20% of all cases. Dull pain and tenderness often accompany bone lesions. Radiographically, the lesions often appear as sharply punched out radiolucencies without a corticated rim, but occasionally an ill-defined radiolucency is seen. Bone involvement in the mandible usually occurs in the posterior areas, and a characteristic „scooped out" appearance may be evident when the superficial alveolar bone is destroyed. The resulting bone destruction and loosening of the teeth clinically may resemble severe periodontitis. Extensive alveolar involvement causes the teeth to appear as if they are „floating in air".

Ulcerative or proliferative mucosal lesions or a proliferative gingival mass may develop if the disease breaks out of bone. Occasionally, this process may involve only the oral soft tissues. Lesions also can occur within the body of the mandible or maxilla, where they may simulate a periapical inflammatory condition.

Histopathologic Features

The bone lesions of patients with Langerhans cell histiocytosis show a diffuse infiltration of large, pale-staining mononuclear cells that resemble histiocytes. These cells have indistinct cytoplasmic borders and rounded or indented vesicular nuclei: Varying numbers of eosinophils are typically interspersed among the histiocyte-like cells. Plasma cells, lymphocytes, and multinucleated giant cells are often seen, and areas of necrosis and hemorrhage may be present.

Treatment and Prognosis

Accessible bone lesions, such as those in the maxilla and mandible, are usually treated by curettage. Low doses of radiation may be employed for less accessible bone lesions, although the potential for induction of malignancy secondary to this treatment is a concern in younger patients. Intralesional injection with cortico-steroids has also been reported to be effective in some patients with localized bone lesions. Infrequently, the apparent spontaneous regression of localized Langerhans cell histiocytosis has been reported. The prognosis for bone lesions in the absence of significant visceral involvement is generally good; however, progression or dissemination of the disease may occur, particularly for patients who have 3 or more bones affected.

4.7. Periodontal Condition in Patients with Down Syndrome and Some other Patological Syndromes

Down syndrome (mongolism, trisomy is a congenital disease caused by a chromosomal abnormality and characterized by mental deficiency and growth retardation. The prevalence of periodontal disease in Down syndrome is high (occurring in almost 100% of patients younger than 30 years old). Although plaque, calculus, and local irritants (e.g.,

diastemata, crowding of teeth, high frenum attachments, and malocclusion) are present and oral hygiene is poor, the severity of periodontal destruction exceeds that explainable by local factors alone.

Periodontal disease in Down syndrome is characterized by formation of deep periodontal pockets associated with substantial plaque accumulation and moderate gingivitis. These findings are usually generalized, although they tend to be more severe in the lower anterior region; marked recession is also sometimes seen in this region, apparently associated with high frenum attachment. The disease progresses rapidly. Acute necrotizing lesions are a frequent finding.

Two factors have been proposed to explain the high prevalence and increased severity of periodontal destruction associated with Down syndrome: a reduced resistance to infections because of poor circulation, especially in areas of terminal vascularization such as the gingival tissue, and a defect in T-cell maturation and in polymorphonuclear leukocyte chemotaxis. Increased numbers of Prevotella melaninogenica have been reported in the mouths of children with Down syndrome.

Chediak-Higashi Syndrome is a rare disease that affects the production of organelles mostly in the melanocytes, platelets, and phagocytes. It is characterized by the partial albinism, mild bleeding disorders, recurrent bacterial infections and rapidly destructive periodontitis.

Hypophosphatasia is a rare familial skeletal disease characterized by rickets, poor cranial bone formation, craneostenosis, and premature loss of primary teeth, particularly the incisors. Patients have a low level of serum alkaline phosphatase, and phosphoethanolamine is present in serum and urine.

Teeth are lost with no clinical evidence of gingival inflammation and show reduced cementum formation.6 In

patients with minimal bone abnormalities, premature loss of deciduous teeth may be the only symptom of hypophosphatasia. In adolescents, this disease resembles localized juvenile periodontitis.

Leukocyte Adhesion Deficiency belongs to rare cases and begin during or immediately after eruption of the primary teeth. Extremely acute inflammation and proliferation of the gingival tissues, with rapid destruction of bone, are found. Profound defects in peripheral blood neutrophils and monocytes and an absence of neutrophils in the gingival tissues have been noted in patients with leukocyte adhesion deficiency; these patients also have frequent respiratory tract infections and sometimes otitis media. All primary teeth are affected, but the permanent dentition may not be affected.

Localized Prepubertal Periodontitis is the form of periodontitis involving only a few teeth and is characterized by minor inflammation and slower bone loss. Mild defects in neutrophils or monocytes, but not both, are found.

Questions

1. What oral and periodontal manifestations of leukemia do you know?
2. What is the difference between the inflamed gingiva in leukemic and nonleukemic individuals?
3. Characterize oral changes in different types of anemias.
4. Characterize periodontal changes in patients with the Immunodeficiency disorders.
5. What are the reasons of the periodontal changes in Diabetes.
6. How is Papillon-Lefevre Syndrome inherited?
7. Enumerate medications used in the treatment of Papillon-Lefevre Syndrome.
8. What are the etiology, clinical signs and prognosis of the

Lipid Reticuloendothelioses?

9. What clinicopathologic disorders are characterized as Histiocytosis X?
10. What are the etiology, clinical signs and prognosis of Hand-Schьller-Christian disease?
11. Describe the Periodontal Condition in Patients with Down Syndrome.

5. NEOPLASTIC ENLARGEMENT (GINGIVAL TUMOURS)

Neoplasms account for a comparatively small proportion of gingival enlargements and make up a small percentage of the total number of oral neoplasms.

5.1. Epulis

Epulis is sometimes a generic term used clinically to designate all discrete tumours and tumour-like masses of the gingiva. Most often it occues in the areas of chronic traumatization. Clinical features are: local gingival enlagement mostly in the area of canines, incisors, premolars. Epulis grows on a broad or narrow peduncular. Its localization is always connected with teeth.

Histopathologically there can be distinguished fibrous, angiomatous and giant cell Epulises.

Fibrous Epulis is firm dark red and consists of connective tissue elements and sometimes osteoblasts.

Angiomatous Epulis is characterized by the intensive growth of blood vesels, which form cavities. It has bluish red appearence, mild consistency, and bleeds readily.

Giant cell epulides is a connective tissue neoplastic enlargement with a large quantity of giant cells. It has greyish blue colour because of the presence of hemosiderin (the result of hemorrhages).

In the initial stages of Epulis the bone underneath is not involved. In the advanced stages Epulides destroys the bone, especially in the interdental spaces. Teeth can be movable because of bone destruction.

Treatment of Epulides is surgical and the coagulation has to be done after the surgery to prevent disease recurrence.

5.2 Fibroma

Fibromas of the gingiva arise from the gingival connective tissue or from the periodontal ligament. They are slow-growing, spherical tumours that tend to be firm and nodular but may be soft and vascular. Fibromas are usually pedunculated. Hard fibromas of the gingiva are rare; most of the lesions diagnosed clinically as fibromas are inflammatory hyperplasias.

Histopathology. The hard fibroma is composed of densely arranged bundles of well-formed collagen fibers with a scattering of flattened elliptical fibrocytes. It is a relatively avascular tumor. In the soft fibroma, fibroblasts are comparatively more numerous and stellate. Collagen is present but is less densely arranged. Various degrees of vascularity are also seen. Bone formation within fibromas is a frequent finding. The bone appears as irregularly arranged trabeculae with osteoblasts and osteoid along the margins. Lipofibroma,

myxofibroma, and peripheral odontogenic fibroma of the gingiva and alveolar mucosa have also been described.

5.3. Papilloma

Papilloma of the gingiva appears as a hard, wart-like protuberance from the gingival surface. The lesion may be small and discrete or may appear as a broad, hard elevation of the gingiva with minutely irregular surfaces.

Histopathology. The lesion has a central core of connective tissue with a marked proliferation and hyperkeratosis of the epithelium.

5.4. Peripheral Giant Cell Granuloma

Giant cell lesions of the gingiva arise interdentally or from the gingival margin, occur most frequently on the labial surface, and may be sessile or pedunculated. They vary in appearance from smooth, regularly outlined masses to irregularly shaped, multilobulated protuberances with surface indentations. Ulceration of the margin is occasionally seen. The lesions are painless, vary in size, and may cover several teeth. They may be firm or spongy, and the color varies from pink to deep red or purplish blue. There are no pathognomonic clinical features whereby these lesions can be differentiated from other forms of gingival enlargement. Microscopic examination is required for definitive diagnosis.

In the past, giant cell lesions of the gingiva have been referred to as giant cell epulides or peripheral giant cell tumors. Most often, however, these gingival lesions are essentially responses to local injury and not neoplasms. When they occur on the gingiva, they should be referred to as peripheral giant

cell granulomas to differentiate them from comparable lesions that originate within the jaw bone (i.e., central giant cell granulomas).

In some instances, the giant cell granuloma of the gingiva is locally invasive and causes destruction of the underlying bone. Complete removal leads to uneventful recovery.

Histopathology. The giant cell granuloma has numerous foci of multinuclear giant cells and hemosiderin particles in a connective tissue stroma. Areas of chronic inflammation are scattered throughout the lesion, with acute involvement occurring at the surface. The overlying epithelium is usually hyperplastic, with ulceration at the base. Bone formation occasionally occurs within the lesion.

Central Giant Cell Granuloma is the lesion which arises within the jaws and produce central cavitation. They occasionally create deformity of the jaw such that the gingiva appears enlarged.

Mixed tumors, salivary gland type tumors, eosinophilic granulomas, and plasmacytomas of the gingiva have also been described but are not often seen.

5.5. Gingival Fibromatosis

Gingival Fibromatosis (Idiopathic Gingival Hyperplasia) has a genetic background as indicated by multiple occurrences in a family. Six of eight children in one family has fibrous hyperplasia (Redman R.S. et al, 1985).

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, where margin and papillae can not be distinguished.

Often even after performed gingivectomy, overgrowth recurred. Sometimes the only way of treatment is teeth extraction and making the complete dentures. Gingival enlargement has a tendency to recur after excision but not after extraction of teeth. Usually no systemic disease can be found in these patients. Gingival fibromatosis patients do not suffer from keloid formation. A few show hirsutism (Horning G.H., et al., 1985) and mental underdevelopment (Willett E., 1954).

5.6. Gingival Cyst

Gingival cysts of microscopic proportions are common, but they seldom reach a clinically significant size. When they do, they appear as localized enlargements that may involve the marginal and attached gingivae. They occur in the mandibular canine and premolar areas, most often on the lingual surface. They are painless, but with expansion they may cause erosion of the surface of the alveolar bone. The cysts develop from odontogenic epithelium or from surface or sulcular epithelium traumatically implanted in the area. Removal is followed by uneventful recovery.

Questions

1. What lesions belong to Neoplastic Enlargement of the gingiva?
2. What are clinical features of the Peripheral Giant Cell Granuloma?
3. How are Gingival Cysts developed?

Test control

1. ANUG does not usually lead to periodontal pocket formation because:
A. Of destruction of junctional epithelium
B. Of rapid gingival recession causing exposure of root surface
C. Of absence of pathogen considered responsible for periodontal pocket formation
D. There is no destruction of tooth supporting structures

2. Punched out, crater-like depressions at interdental papillae as seen in ANUG, may also be observed in:
A. HIV-associated gingivitis
B. HIV-associated periodontitis
C. Streptococcal gingivostomatitis
D. Desquamative gingivitis

3. HIV associated periodontitis has been found to be strongly associated with:
A. Subgingival Candida albicans
B. Kaposi's sarcoma
C. Recurrent herpes simplex virus
D. Pneumocystis carinii pneumonia

4. A compound periodontal pocket is:
A. Spiral type of pocket
B. Present on two or more tooth surfaces
C. Infrabony in nature
D. All of the above

5. Normally, the distance between the junctional epithelium and the alveolar bone:
A. Increases with age
B. Decreases with age

C. Remains constant
D. Increases in periodontal disease

6. The transition from gingivitis to periodontitis:
A. Occurs when there is a shift in microflora from motile rods to filaments type
B. Is always observed in longstanding cases
C. Is accompanied by severe pain and induration
D. Is the result of extension of inflammation into periodontal tissues

7. During surgical procedures for pocket eradication, the firm covering over the bone encountered after the superficial granulation tissue is removed, is:
A. Periosteum
B. Trans-septal fibres
C. Densly organised granulation tissue
D. Cartilaginous tissue

8. Traumatic occlusion' is synonym of:
A. Trauma from occlusion
B. Malocclusion
C. Traumatism
D. None of the above

9. Localised juvenile periodontitis is usually observed in:
A. Down syndrome
B. Papillon'-Lefevre syndrome
C. Both of the above
D. None of the above

Epidemiology and Pathology

10. Juvenile periodontitis:
A. Is a degenerative disease
B. Affects males more than females
C. In its early stage, shows lack of clinical inflammation in presence of deep periodontal pockets
D. Is self-limiting in nature

11. Periodontal index (PI) by Russell:
A. Considers all of the tissue circumscribing a tooth, as one scoring unit
B. Measures only the reversible aspect of periodontal disease
C. Overestimates the true level of periodontal disease, especially early bone loss
D. Follows same criteria for field and clinical tests

12. The score of 8.0 in Russell periodontal index (PI) indicates:
A. Irreversible terminal disease
B. Established destructive periodontal disease
C. Beginning of destructive periodontal disease
D. PI score is always less than 1

13. Simplified oral hygiene index by Green and Vermillion:
A. Stipulates scoring on each tooth present in oral cavity
B. Gives a score of '2' for the presence of individual flecks of subgingival calculus around the cervical portion of the tooth
C. Scores for both plaque and calculus
D. Is cumbersome and difficult to interpret

14. Community periodontal index of treatment needs (CPITN):
A. Was developed by American Dental Association in 1982

B. Is scored by a special periodontal probe having its length between 3 and 5 mm blackened for easy visibility
C. Measures '6' representative teeth in epidemiologic surveys
D. Score of 3' indicates treatment which includes oral hygiene instructions and scaling only

15. The area of oral cavity most severely affected by gingivitis is:
A. Interproximal, upper arch
B. Interproximal, lower arch
C. Buccal, upper arch
D. Lingual, lower arch

16. Human immunoglobulin is divided into five classes (IgA, IgG, IgM, IgE and IgD) on what basis:
A. Functional differences
B. Structural differences
C. Antigenic affinity
D. Complement fixation

17. The principal inorganic components of the supragingival plaque matrix are:
A. Calcium and phosphorus
B. Calcium and magnesium
C. Calcium and potassium
D. Phosphorus and magnesium

18. Subgingival calculus:
A. Cannot occur without supragingival calculus
B. Contains less hydroxyapatite than supragingival calculus
C. Is uncommon in children
D. Derives its minerals from same source as supragingival calculus

19. Calculus:
A. Per se is the irritating cause to gingiva
B. Is always covered with a non-mineralised layer of plaque
C. Is formed as all plaque undergo mineralisation
D. Formation cannot be obtained in germ-free animals

20. Orange stains, seen on both facial and lingual surfaces of anterior teeth are produced by:
A. Aspergillus
B. Serratia marcescens
C. Bacteroides melaninogenicus
D. None of the above

21. The location of margins of a restoration associated with periodontal health is:
A. Subgingival
B. At the level of gingival crest
C. Supragingival
D. Varying depending upon type of restoration

22. Bruxism can occur as:
A. Clenching of teeth
B. Grinding of teeth
Q. Rhythmic side-to-side movements
D. All of the above

23. Bruxism:
A. Is easy to diagnose
B. Is stress-related in some individuals
C. Is usually hereditary
D. Can be controlled by extensive occlusal adjustment

24. Pregnancy-related gingivitis:
A. Is associated with significant increase in Bacteroides gingivalis levels
B. Is most prominent in II trimester of pregnane)
C. Both of the above
D. None of the above

25. In acute leukemia, microscopically gingiva shows:
A. Leukemic cell infiltration in epithelium
B. Inflammatory cell infiltration in all connective tissue
C. Intercellular and intracellular oedema in epithelium
D. All of the above

26. Gingival abscess:
A. Is confined to marginal gingiva
B. Is associated with chronic gingivitis
C. Usually occurs in children
D. All of the above

27. Chronic periodontal abscess:
A. Is usually associated with continuous dull, gnawing pain
B. Is located on the same surface of the root as the pocket from which it forms
C. Usually present a sinus opening onto the gingiva along the length of root
D. All of the above

Diagnosis and Treatment

28. Gracey curettes:
A. Can work with either cutting edge
B. Are curved in two planes
C. Have their blade angled approx 80 degrees to lower shank
D. All of the above

29. The flap technique for pocket elimination which also increases width of attached gingiva is:
A. Coronally repositioned flap
B. Apically repositioned flap
C. Laterally displaced flap
D. Modified Widman flap

30. The unrepositioned flap:
A. Eliminates pocket wall
B. Is also considered as internal bevel gingivec-tomy
C. Can be performed only if sufficient width of attached gingiva is present
D. All of the above

31. In modified Widman flap:
A. Internal bevel incision is the last incision
B. Interdental papilla is eliminated
C. Posterior areas are difficult to access
D. Crevicular incision is made from base of the pocket to the bone

32. Dental floss:
A. Is used to remove food wedged in between two teeth
B. Is moved to and fro in a facio-lingual direction in the interproximal area
C. Is difficult to use in gingival embrasures occupied by intact papillae
D. None of the above

33. The minimum concentration of chlorhexidine, required to be effective as a plaque inhibitor is:
A. 0.22%
B. 0.20%

C. 0.12%
D. 0.10%

34. The minimum time for which chlorhexidine should be kept in oral cavity during rinsing, for the effective plaque control is:
A. 10 sec
B. 20 sec
C. 30 sec
D. 40 sec

35. During scaling on heavy calculus, the angulation of the instrument should be:
A. Less than 45"
B. Slightly less than 90°
C. Slightly more than 90°
D. Slightly more than 45"

36. Regeneration of lost periodontium involves:
A. Healing by new attachment formation
B. Healing by longjunctional epithelium formation
C. Healing by re-attachment
D. Healing by scar formation

37. Xerostomia in aged patients may be successfully treated with:
A. Artificial saliva substitutes
B. Equal parts elixir of diphenhydramine and Kaolin, 1 minute oral rinse
C. 2 % lidocaine viscous mouthwash
D. All of the above

38. Dental calculus contains:
A. Vital microorganisms
B. Non-vital microorganisms

C. Both of the above
D. None of the above

Miscellaneous

39. The number of bacteria in the oral cavity is greater:
A. In the morning
B. After meals
C. At night
D. After burshing

40. The predominant organism found in smears of ANUG is:
A. Vibrio
B. Fusobacterium
C. Spirochaetes
D. Streptococci

41. Vincent's angina can be differentiated from Vincent's stomatitis as:
A. It involves the oropharynx and throat whereas latter does not
B. Both are same
C. It is a fusospirochaetal infection whereas latter is caused by streptococci
D. Former is a painless condition and latter is very painful

42. A case presenting with a grey colored pseudo-membrane whose removal is difficult and painful, can be:
A. ANUG
B. Diphtheritic lesion
C. Secondary stage of syphilis
D. Desquamative gingivitis

43. A patient presents with a pseudomembrane on the gingiva which cannot be peeled off; bacterial smear shows spirochetal infestation and it appears to respond to antibiotics. It can be:
A. ANUG
B. Diphtheritic lesion
C. Secondary stage of syphilis
D. Desquamative gingivitis

44. A patient had undergone restoration of 1st molar. 2 days after operative procedure, patient developed shiny erythema with numerous pinpoint vesicles confined to mucogingival area of operative site. Later vesicles ruptured, forming painful ulcerations. 7-10 days later uneventful healing occurred. It could have been:
A. Pressure sore
B. ANUG
C. Acute herpetic gingivitis
D. Bullous lichen planus

45. A 36-year-old female patient presents with extensive vesicles formation over gingiva, lips, cheeks and tongue. 2-4 days later the vesicles rupture to form pseudomembrane. There are few skin lesions also present; the condition can be:
A. Acute herpetic gingivitis
B. Erythema multiforme
C. ANUG
D. Stevens Johnson syndrome

46. The lesions seen in apthous stomatitis, as compared to herpetic gingivostomatitis are:
A. Larger
B. Smaller
C. Less discrete
D. More diffuse

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