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INFLAMMATORY PERIODONTAL DISEASES: PATHOGENESIS, PRINCIPLES OF COMPLEX TREATMENT

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ABSTRACT

The article presents modern ideas about the features of the occurrence and course of gingivitis and periodontitis, taking into account the hormonal, immune and age status of the patient. An algorithm of therapeutic measures and medication is presented, including the use of extemporaneous paste BKM and medicinal films developed by the author.

KEY WORDS: *Periodontitis , Periodontitis Treatment , Medicinal Films, Extemporaneous Paste*

PATHOGENESIS

Inflammatory periodontal diseases are one of the most complex problems in dentistry, which is explained by both its multicomponent nature and the persistence of the course due to the insufficient effectiveness of the proposed means and methods of their application. From the moment of teeth eruption, microorganism aggression unfolds in the oral cavity, to which the macroorganism responds by turning on numerous protective mechanisms. These include immune factors secreted into the gingival fluid and saliva, and the migration of normal, very active macrophages and the sufficiently intensive blood supply that ensures their entry into the arena of action, and the rapid change of the integumentary epithelium, and the extremely intensive process of collagen regeneration. On the side of bacteria, there are such damaging factors as thermal (temperature changes from the lowest to barely bearable high), and mechanical damage when using too coarse food and malocclusion, and chemical aggression in acute and chronic poisoning with heavy metals. The process is aggravated by insufficiently effective or completely ignored oral hygiene by the patient. Deeper development of the process with loss of calcium from the hard tissues of the dental and jaw system is often associated with hormonal disorders combined with vitamin imbalance. This long-term process develops against the background of the slow but sure aging process of the body.

It is recognized that the initially vulnerable link is the gingival groove, adjacent to the enamel and covered with transitional epithelium. Directly adjacent to it is a dense vascular plexus. Through the walls of its capillaries there is a constant flow of polymorphonuclear neutrophil granulocytes (NG), which , passing through the transitional epithelium layer, end up in the gingival sulcus fluid and then in the oral cavity. Healthy and full-fledged NG accumulate and function between the gum tissue and microbial plaque, forming a protective barrier. This leads to the limitation of the spread of microbial plaque. The second line of defense against bacterial aggression are fibroblasts of the connective tissue of the gums, supporting and renewing the fibrous matrix of the connective tissue. At the same time, near the blood vessels are and mast cells, which are a reservoir of inflammatory factors, which at first also have a defensive function.

At some stage, as a result of primary damage, microcirculation in the gums is disrupted, pO2 in the tissues decreases, which leads to a short-term spasm with a longer-term vasodilation . This is manifested in an increase in vascular permeability with the penetration of plasma proteins into the walls of blood vessels, and then perivascularly .

These metabolic disorders can arise as a result of a banal deficiency of ascorbic acid or as a manifestation of diabetes. The collagenase activated in this case leads to depolymerization of collagen against the background of weakening of fibroblast function. Disorganization of connective tissue occurs due to the breakdown of hyaluronic acid against the background of lohypoxia . Vasculitis develops , and local accumulation of PNH constitutes a pathological syndrome of capillary-trophic insufficiency.

Over time, the damage expands, the number of plasma cells increases, and an initial gingival pocket is formed. It is filled with migrants - PNG, which go further - into the oral cavity. Initial signs are most often observed in the area of the gingival groove in the interdental spaces.
Such damages – of moderate and even moderate severity – can disappear spontaneously, more often – remain stable or turn into a destructive process with alteration of the gum edge and resorption of the alveolar bone.

Chronic periodontitis is characterized by the presence of gingival pockets with ulceration of the epithelium lining the pockets, the formation of a dense infiltrate of lymphocytes, plasma cells, macrophages and a variable number of neutrophils. At the same time, there is a decrease in the amount of collagen with fibrosis, destruction of the normal structure of the connective tissue adjacent to the root of the tooth, as well as osteoclastic resorption and destruction of the alveolar bone and root of the tooth with a cyclical or disordered course of the process.

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The described damage to connective tissue and periodontal ligament occurs against the background of a reduction in periodontal collagen with a simultaneous increase in its synthesis – with the formation of type J collagen and weakly intertwined soluble collagen.

In accordance with these pathological changes in the matrix of connective tissue of the gums, both the transitional epithelium and the epithelium of the pockets are greatly weakened, the connection between the tissues of the gum and the tooth is broken, and the epithelial barrier is lost, which further increases inflammation.

From this point on, the growth and development of subgingival bacterial plaques increases. The transitional epithelium shifts in the apical direction, which correspondingly deepens the pocket. Bacteria now begin to actively penetrate the periodontal tissues, causing a further response from the host. With timely removal of the bacterial factor, inflammation subsides: the number of neutrophils and other "inflammatory cells" is significantly reduced, the structure of the connective tissue, the epithelial barrier, including the transitional epithelium, is restored.

Epithelium.
Clinic. Inflammatory periodontal diseases - gingivitis and periodontitis are a single process that developed gradually in the early years (from childhood to 25-30 years) and does not yet lead to a violation of the tooth-gingival junction, which is considered as gingivitis of varying severity. With the appearance of a periodontal pocket, the stage of the process is considered as periodontitis, in which deeper (in anatomical and functional terms) changes occur.

Gingivitis is an initially localized and later generalized inflammation of the mucous membrane of the alveolar process of the gum. It is characterized by the presence of soft (non-mineralized) plaque, food particles and, rarely, tartar. The severity of the process in the gum directly

proportional to the degree of oral hygiene. Often a combination of gingivitis with caries in the spot stage (focal demineralization of enamel) in the cervical area. As a rule, swelling and inflammatory infiltration of the gums are observed, sometimes with signs of proliferation in the area of the gingival papillae. Increased bleeding and pain upon contact with the gum are typical. The general condition of patients is not disturbed, except for cases of general intoxication of patients.

With gingivitis of any severity, there is no periodontal pocket, tooth mobility, or signs of alveolar bone resorption.
Severity of gingivitis: mild gingivitis is characterized by damage to the interdental gums, moderate gingivitis is characterized by damage to the interdental and marginal gums, and severe gingivitis is characterized by damage to the entire gums, including the alveolar gums.

Localized gingivitis is recognized if the gum is affected in the area of 1-2-3 teeth. Generalized gingivitis is characterized by damage in the area of all teeth of the upper and lower jaw. In the clinic, chronic catarrhal gingivitis in the acute stage is most often observed. In some cases, acute catarrhal gingivitis is accompanied by serous inflammation of the entire mucous membrane of the oral cavity, and then it is regarded as gingivostomatitis .

Acute catarrhal gingivitis : severe swelling of the gums causes the epithelium to be shiny and tense, accompanied by pain and bleeding when touched. Acute catarrhal gingivitis often occurs in acute intoxication (with bismuth, mercury, and lead salts).
Chronic catarrhal gingivitis usually causes little concern to patients; they rarely seek medical attention due to the weak severity and inconsistency of the symptoms of inflammation.

Chronic catarrhal gingivitis is often accompanied by chronic diseases such as liver cirrhosis, chronic glomerulonephritis , thyrotoxicosis and myxedema, as well as hypovitaminosis C.

Periodontitis is the next stage of gum inflammation, when the process affects the underlying periodontal tissues. It is characterized by progressive destruction of the periodontium and bone structures of the interdental septa. This is usually detected at the age of 30-40 years.

Characteristic signs of periodontitis: the presence of a periodontal pocket and, in addition to plaque, significant deposits of supragingival and subgingival tartar; tooth mobility, displacement, impaired chewing function, tremas , traumatic articulation. Radiographs show signs of tissue destruction of the interdental septa with the destruction of the cortical plates. Foci of spotted osteoporosis and blurred contours of the septa are noted.
Patients show signs of general malaise, elevated body temperature, changes in the peripheral blood picture according to the type of non-specific inflammation - leukocytosis, increased ESR.

Localized periodontitis can develop in the area of 1-2-3 teeth due to occlusal trauma, rupture of the circular ligament during prosthetics, introduction of filling material into the interdental space. With poor oral hygiene, the localized process can transition to a generalized form. With timely elimination of the causative factor, the prognosis is favorable - with possible regeneration of bone tissue. Generalized periodontitis is an inflammatory lesion of all periodontal tissues. It is characterized by heterogeneity of the periodontal tissue lesion: periodontal pockets of varying depth, and the degree of mobility of individual teeth is also different. Even within one tooth, bone resorption is uneven. This leads to early removal of individual teeth and the formation of traumatic occlusion. The course of the disease is progressive, with frequent exacerbations, abscess formation . Abscesses and fistulas are located closer to the gingival margin against the background of edematous and hyperemic mucosa. At the same time, the general condition suffers - headache, temperature reaction with general malaise are observed. This leads patients to see a specialist. With active complex treatment using surgical

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intervention, stabilization of the process with partial regeneration of bone tissue is possible.

The severity of the process is determined by the depth of periodontal pockets and the degree of bone tissue resorption. This determines the amount of tooth mobility, as well as the choice of surgical and orthopedic treatment tactics.
A mild degree of periodontitis is characterized by the following indicators:
periodontal pocket depth up to 3.5 mm;
– no loose teeth;
– no displacement of teeth;
- induced bleeding;
– the general condition of the patient is not disturbed;
- radiologically: disintegration or disappearance of the compact plate;
– reduction of interdental septa to less than 1/3 of their height.
The average degree of periodontitis is characterized by the following indicators:
periodontal pocket depth up to 5.5 mm;
– pathological mobility of teeth of 1-2 degrees;
– there is displacement of teeth, tremas , diastemas ;
– traumatic occlusion appears;
– resorption of interdental septa from 1/3 to ½ of their height;
Severe periodontitis is characterized by:
– periodontal pockets more than 6 mm;
– teeth mobility of 2-3 degrees;
– teeth are displaced;
- significant tremes ;
- pronounced traumatic articulation;
– there are defects in the dental arches;
– resorption of tissue of interdental septa from ½ the length of the root to complete.
In addition, with moderate and severe periodontitis
purulent discharge from periodontal pockets and abscess formation are possible .
In the remission phase of periodontitis the following is typical:
– the gums are pale pink, tightly adjacent to the surface of the tooth;
– possible exposure of the tooth root;
– the depth of the periodontal pocket can be maintained
sya , but with some types of surgical treatment it may
decrease to 1-2 mm with light probing or be absent;
- there are no radiological signs of active progression
process – new foci of osteoporosis are not visible, no progressive loss of bone tissue is observed;
– chewing function, diction and aesthetics improve.

A characteristic anamnesis, which confirms the implementation of complex treatment and other anti-inflammatory measures, helps in establishing the diagnosis of “periodontitis in remission”.

Typical complaints with advanced periodontitis: bleeding gums, pain when biting, sticky plaque on the teeth, bad breath (often "sweetish"), the appearance of severe pain deep in the jaw, lasting for several minutes. During such an attack, percussion on the teeth does not change nature of pain. Sometimes the only complaint is an increase in the gaps between teeth that have become mobile or the release of pus from under the gum.

Treatment. A modern comprehensive approach to the treatment of inflammatory periodontal diseases should become the realization of the idea of great doctors: "treat the patient, not the disease."

This involves a thorough examination of previous and current suffering, consideration of the patient's hormonal and age status, medications previously used and currently used, allergy susceptibility, vitamin balance and possible genetic predisposition. This information should be contained in the patient's medical record and, ideally, in the patient's electronic medical history.

Taking into account the above, oral hygiene should be in the first place in terms of prevention and therapy. This will significantly reduce bacterial aggression, which has often been unsuccessful when using only pharmacological antibacterial drugs: they may be ineffective in the presence of antibiotic - resistant strains. In addition, the bactericidal effect does not at all mean the exclusion of the effect of bacterial exotoxins remaining in the tissue. And in the case of anaerobic microflora - also endotoxins, which are especially harmful to the deep sections of the periodontium and especially its bone base.

A comprehensive therapy for inflammatory periodontal diseases should be built taking into account the main links in pathogenesis and the essence of the disease process:
– the presence of diverse flora in the form of plaque, plaque and hard dental deposits;
- the adhering fungal flora is insensitive to antibacterial agents;
– inflammatory tissue swelling;
– deterioration of microcirculation and pO2 deficiency ;
– decrease in the production of complete collagen;
– the release of calcium from the protein matrix;
– the presence of excess pathological granulation in the pockets;
– shift of pH towards the acidic side.
oral hygiene comes first, both during the period of prevention and treatment of this pathology. It includes mechanical, including the use of ultrasound, removal of tartar, bacterial plaques and excess granulation.

In the presence of periodontal pockets, the first stage is their curettage followed by sanitation of the surfaces with a 0.06% chlorhexidine solution . In the case of a more widespread and deep process, the second stage should be carried out - a flap operation, which is necessarily carried out under conduction anesthesia (application anesthesia is insufficient both in depth and duration). Upon completion of the procedure, the flap is pressed tightly to the surface of the tooth, leaving blood in place , which is organized into a blood clot as a source of epithelial cells for the formation of a tooth-gingival junction close to normal .

Better fixation of the flap is also facilitated by the use of gingival dressings and the reduction of tooth mobility by their

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temporary application . splinting . In case of using medicinal preparations, their effectiveness depends on the duration of their contact with tissues. In order to increase this indicator, it is advisable to use medicinal films or extemporaneous pastes containing a complex of preparations. In case of using medicinal preparations, their effectiveness depends on the duration of their contact with tissues. Longer contact is achieved by using extemporaneous pastes with increased adhesion, as well as medicinal films. The complex of preparations included in their composition has antibacterial, fungicidal , anti-inflammatory, anabolic and calcium-sparing effects.
Deeper penetration of the paste components is ensured by the inclusion of dimethyl sulfoxide (DMSO), which has some analgesic effect.

Briefly, these manipulations can be represented as follows:
Ulcerative gingivitis:
- after anesthetizing the gums, soft dental plaque and hard dental deposits are removed, then - applications of enzyme preparations (chymopsin , trypsin or chymotrypsin) diluted in an isotonic solution of sodium chloride or distilled water. After mechanical cleaning of the ulcer surfaces from necrotic masses and antiseptic treatment of the gums, BKM paste is applied to the affected areas of the gums on sterile gauze strips for 20 minutes, after isolating it from saliva. At home, it is recommended to carry out antiseptic mouth rinses every 1.5-2 hours, hygienic care with a soft toothbrush 3-4 times a day. Anti-inflammatory and antimicrobial drugs are prescribed internally (depending on the severity and taking into account the indications) (co- trimoxazole 480 1 tablet 2 times a day, metronidazole 1 tablet 3 times a day) for 7-10 days, and after eliminating inflammation in the gum - drugs that help increase the body's natural resistance (methyluracil 1 tablet 3 times a day for 10-15 days), vitamin therapy (vitamin "A" in oil locally in the form of applications on the gums, Aevit 1 capsule 3 times a day, Ascorutin 1 tablet 3 times a day, etc.).
Generalized periodontitis: - local therapy begins with oral cavity sanitation (removal of dental plaque, treatment of carious teeth, removal of roots of teeth and prostheses that do not represent functional value, etc.). Depending on the indications, selective grinding of teeth (to create multiple uniform contacts, eliminating overload of periodontal tissues) and temporary splinting to stabilize loose teeth. As a result of orthopedic treatment methods, hemodynamics and metabolic processes in the periodontium are normalized, the resistance of periodontal tissues to mechanical stress and the effects of unfavorable factors increases. Removal of dental plaque should be carried out very carefully using modern ultrasonic devices.

Local treatment of periodontal tissues consists of 3 stages:
1) drug therapy, the purpose of which is to eliminate pathogenic microflora and eliminate inflammation in the gums;
2) surgical treatment, the purpose of which is to eliminate the periodontal pocket (curettage – removal of subgingival dental deposits, granulations, altered cementum, epithelial strands from the inner surface of the gum; flap surgery, method of guided regeneration, etc.)
3) postoperative stage until the patient is fully rehabilitated.

When treating with BKM paste, at the first stage of treatment it is necessary to adhere to a strict sequence of therapeutic interventions on the periodontium: after removing dental plaque and irrigating the pockets from a syringe under pressure with antiseptic solutions, drying the area of the affected periodontium with a stream of compressed air, it is necessary to introduce BKM paste into the periodontal pockets under the gingival dressing. The procedure is repeated until the inflammation in the gum is completely eliminated. However , in some cases, the inflammatory reaction stubbornly persists. This is explained by the fact that chronic inflammation significantly increases the activity of physiological mechanisms that play a protective role and ensure the viability of tissues in conditions of impaired hemodynamics and the action of pathogenic factors. Therefore, if inflammation in the gum persists, then it is necessary to continue treatment with BKM paste until the inflammatory reaction is completely eliminated. The method of using a particular drug is of no small importance. In case of minor exudation from pockets, BKM paste can be introduced into periodontal pockets under the gingival medicinal and protective layer in order to prolong its action.

Bandage. In case of abundant suppuration, the use of bandages is excluded in order not to cause the formation of a periodontal abscess. For the same reason, active drug therapy in the form of irrigation of pockets from a syringe under pressure, the introduction of BKM paste into periodontal pockets under

Gum dressings are not used in the case of abscessed periodontitis, severe exudation from pockets, and symptoms of general intoxication of the body. Applications of BKM paste are prescribed 2-3 times a day with an exposure of 20 minutes. After the exacerbation has been eliminated, treatment is carried out according to the above-mentioned method and averages from 2 to 4 procedures, depending on the severity of periodontitis.
In the postoperative stage, until the patient is fully rehabilitated, it is possible to use BKM paste as part of therapeutic and protective gum dressings.

The described procedures on the periodontium should be supported by the resorptive effect of a complex of drugs - vitamins of targeted action, non-steroidal anabolics, inhibitors of bone resorption, factors promoting remineralization . The direction of the effects of drugs of resorptive use
Vitamins:
C (ascorbic acid) – participates in oxidation-reduction reactions, improves protein synthesis (including collagen), participates in the synthesis of steroid hormones, increases the resistance of capillaries (in cooperation with vitamin P – rutin).
B 1 – participates in carbohydrate metabolism, reducing the level of pyruvate , increases the energy supply of tissues.
B 2 - participating in protein, fat and carbohydrate metabolism, prevents the development of cheilitis , gingivitis. B 6 - participates in protein metabolism, transamination of tryptophan, methionine, cysteine, glutamine, thereby strengthening the protein matrix of the gums .

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D- ergocalciferol ensures the absorption of calcium from food and reduces its loss through the kidneys. Non-steroidal anabolics:
Methyluracil – has an anti-inflammatory effect, improves nucleic acid metabolism and regeneration in bone pathology and ulcerative processes.

Orotate is an anabolic that does not cause calcium loss and stimulates regeneration and protein synthesis.
Bone resorption inhibitors : K alcitonin - a preparation of the natural hormone calcitrin (obtained by genetic engineering) is an analogue of a bone mineralization stimulator. 1 ml contains 50 IU.

Xidifon (etidronic acid) is an inhibitor of osteoclastic bone resorption.
Osteogenon is an ossein- hydroxyapatite compound. It is an osteoblast stimulator and an osteoclast inhibitor.
Remineralization preparations : calcium carbonate, calcium gluconate, calcinate (a complex of retinol , ergocalciferol , pyridoxine, calcium) and a complex of vitamins A , D, B6 with calcium phosphate. The duration of the achieved
This therapeutic effect is also determined by the formation of a stable motivation of the patient for regular and properly performed oral hygiene.

10. 2 p.

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