METHODS OF TREATMENT OF GENERALIZED AGGRESSIVE PERIODONTITIS

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Annotation*. Aggressive periodontitis is a type of periodontitis characterized by lightning-fast destruction of the periodontal ligament and loss of alveolar bone mass and occurs, as a rule, in young people, less often in elderly patients [1]. According to the classification of periodontal diseases adopted at the meeting of the Presidium of the Periodontology Section of the Dental Association of Russia in 2001, this disease was allocated to an independent group of periodontal diseases - aggressive forms of periodontitis. However, dentists more often turn to the classification of dental diseases ICD-C-3 (ICD-C-3), based on ICD-10 (WHO, 1995), according to which aggressive periodontitis corresponds to the code k 05.4 "periodontitis". A population of 267 patients (165 men (60.3%) and 102 women (39.7%)) was studied at our department it was 89 cases (1.65%). No significant differences were found between the percentage of male and female patients. The generalized form was more common than the localized one, in a ratio of 27:1.*

Key words: *diseases, radiation, treatment, diagnostics.*

УМУМИЙ АГРЕССИВ ПЕРИОДОНТИТНИ ДАВОЛАШ УСУЛЛАРИ

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Аннотация*. Агрессив периодонтит-бу перидонтал лигаментнинг яшин тезлиги билан тез парчаларишu ва алвеоляр суяк массасининг йўқолиши билан тавсифланган периодонтитнинг бир тури бўлиб, бу касаллик асосан ёшларда, камроқ кекса беморларда учрайди [1]. 2001 йилда Россия стоматология Ассоциациясининг Периодонтология бўлими Президиумининг йиғилишида қабул қилинган перидонтал касалликлар таснифига кўра, бу касаллик перидонтал касалликларнинг мустақил гуруҳига - периодонтитнинг агрессив шаклларига ажратилган. Ўтказган илмий тадқиқотларимиз асосида, 267 нафар беморларнинг ўрганиш аҳоли агрессив парадонтит частотаси (165 эркаклар (60,3%) ва 89 аёллар (39,7%)) бу 76 ҳолатлар (1,65%) эди. Эркак ва аёл беморларнинг улуши ўртасида сезиларли фарқлар топилмади. Умумлаштирилган шакл локализацияга қараганда 27:1 нисбатда кенг тарқалган эди.*

Калит сўзлар*: касалликлар, нурланиш, даволаш, диагностика.*

The average age was 34.5 years [2]. Although the prevalence of aggressive periodontitis is much lower than in other periodontitis diseases, such an invariable risk factor as a strong genetic predisposition causes significant difficulties in treatment. The absence of early diagnosis and timely therapy in patients with this disease in a short time can lead to rapid destruction of periodontal tissues and tooth loss [3]. There are specific diagnostic signs, but their clinical manifestations may vary in different patients. If the disease is diagnosed at later stages, then successful treatment is a challenge. To date, there is a wide range of available treatments with fairly variable success rates. However, research in this area is increasing exponentially, and there are treatment options such as

- Tissue engineering and genetic technologies are promising in relation to this disease In case of a collision with this pathology in clinical practice, it is necessary to take into account that generalized aggressive periodontitis is a multifactorial disease in which microbiological, genetic, immunological and behavioral factors are integrated. They determine the onset, course and severity of the disease. Bad habits, such as smoking or poor oral hygiene in patients, cause a greater destruction of periodontitis in comparison with those who maintain the condition of the oral cavity at a satisfactory level.

The disease has a wavy course with periods of exacerbation and remission. This leads to two different clinical pictures at the time of the examination. During remission, patients do not make any complaints, the gums are pink, but when probing, deep periodontal pockets are detected. The lack of visible signs of an inflammatory reaction is a characteristic feature of aggressive periodontitis, which is detectable at this stage of the course of the disease, despite deep temporomandibular joints and disorders of the gum connection, along with complete somatic health and the young age of patients. Periods of remission can last from several weeks to several months or years, accompanied by periods of exacerbation of the disease. At the stage of exacerbation, progressive destruction of the alveolar bone and loss of the temporomandibular joint occurs. During this stage, during a clinical medical examination, the gums have all the signs of inflammation, ranging from mild to severe severity. Often there is an inflammatory gingival

hypertrophy. When probing with a calibration probe, it is characterized by bleeding in six areas around each tooth or even spontaneous release of purulent exudate. Most patients turn to a dentist at this stage of the disease.

The prognosis of treatment largely depends on the timely diagnosis. Early diagnosis of the disease helps to prevent its progression and avoid severe mass loss of the alveolar bone. In addition, since aggressive periodontitis is prone to a family predisposition, it is necessary to examine close relatives in order to diagnose the disease in other family members at an early stage [4].

To date, conservative treatment remains the main method of antibacterial therapy for generalized aggressive periodontitis. At the initial stages of the disease, when the destruction of the periodontal joint and bone is mild to moderate, the treatment of the disease is based on the use of systemic antibiotics in combination with mechanotherapy. Treatment should begin with an attempt to completely eliminate the bacteriostatic effect on etiological factors and modifiable risk factors. It should be remembered that the disease has a strong genetic predisposition. In its pathogenesis and manifestation, an important role is played by the reaction of the human body to the pathogenic microorganisms of plaque. This response is determined genetically and is an invariable risk factor for the development of the pathology in question [5]. However, the disease also depends on the microbial factor and environmental factors, so it can be successfully monitored in susceptible people, which indicates that it is absolutely important for the patient or the doctor to remove dental deposits at home at an early stage at a dentist's appointment. In patients prone to this disease, even a small amount of plaque can trigger a reaction to humoral and cellular immunity. The mechanical removal of plaque from the tooth surface can be achieved by training and motivating the patient to proper oral hygiene by vividly demonstrating the modified Bass method (for patients without gum recession) and the modified Stillman method (for patients with hyperesthesia) on the tooth cleaning model. Also, the use of hygiene aids for cleaning the interdental gaps (dental floss, interdental brushes, toothbrushes) provides effective treatment of the hard-to-reach areas of the oral cavity. Systematic visits to the dentist are of great importance for controlling the effectiveness of plaque control. For further control, in addition to mechanical measures, a mouth rinse with 0.12% or 0.2% chlorhexidine solution or a 1% povidone iodide solution can be recommended [6]. Aminofluoride and tin fluoride, consisting of dishwashing detergents and toothpastes, are also effective in combating supraplaques in aggressive periodontitis. In addition, the use of fluoride for rinsing the oral cavity is recommended for remineralization of open root surfaces, as well as as a desensitizing agent in patients with complaints of increased tooth sensitivity [7]. It has been scientifically proven that smoking is a significant risk factor and leads to a more severe course of the disease. The effectiveness of conservative and surgical treatment, restorative therapy in such individuals is much lower than in non-smoking patients. Therefore, at an appointment with the dentist, the doctor should inform about the effects of smoking on the periodontal condition and the benefits of refusal [8Systemic antibacterial therapy. The use of systemic antibiotics is an integral part of the treatment of aggressive periodontitis, since pathogenic bacteria such as Aggregatibacter actinomycetem comitans and Porphyromonas gingivalis cannot be completely destroyed by other methods [16]. Previously, the antibiotics of the tetracycline group were widely used to achieve this goal. They have the ability to concentrate in the periodontal tissues and inhibit the growth of A. Actinomycetem comitans. In addition, tetracyclines inhibit collagenase, reduce tissue destruction and promote bone regeneration [17]. However, the development of resistance to them led to the use of other pharmacological groups as combined or sequential therapy. Currently, it is preferable to use a combination of amoxicillin three times a day for 250 mg together with metronidazole twice a day for 250 mg for 8 days. This is one of the most effective combinations of drugs, which has sufficient evidence for this [18]. In the case of therapeutic treatment at the initial stages of the disease, antibacterial therapy significantly improves the results of treatment as a whole. The identification of bacterial species and their sensitivity to antibiotics is not an obligatory measure, since the combinations described above can be more clinically and cost-effective. The use of only one drug (doxycycline, azithromycin, metronidazole and clindamycin) gives results when used in addition to SRP. The choice of an antibiotic is carried out taking into account numerous criteria - possible allergic reactions on the part of the patient, concomitant pathologies, anamnesis data [19].

Local use of antimicrobial drugs is also a necessary component of the therapeutic treatment of aggressive periodontitis, mainly with limited accumulation of exudate, deep periodontal pockets and lack of adequate response to mechanical therapy and the use of systemic antibiotics. The main advantage of this method is to put smaller doses of drugs in the dental pocket, maintaining the concentration of the active substance in the focus of infection at a high level. This avoids the side effects typical of systemic antibacterial agents.

In addition, this option is applicable for intolerance to systemic administration of drugs. Recently, drugs with a gradual and slow release of the active substance have been used. The decision on the use of local anti-infection therapy remains subject to individual clinical evaluation, the treatment phase, as well as the status and selection of the patient [20].

A review of the literature and scientific studies on the topic of non-surgical treatment of generalized aggressive periodontitis has shown that this pathology can be successfully treated with therapeutic methods. Since such treatment stops the progression of the disease, relieves inflammation, reduces the depth of periodontal pockets, significantly reduces the reconstruction of alveolar defects. Based on these data, the treatment plan should begin with basic or complementary methods of mechanical antimicrobial therapy in combination with systemic antibiotics. It is recommended to start antibacterial therapy 24 hours before SRP or other methods of mechanical removal of dental deposits and continue these procedures in parallel. After 4-6 weeks, the condition of the patient's oral cavity is assessed again. If there are no significant changes at the initial stages of therapy, the doctor may repeat the treatment with various combinations of antibiotics and possible treatments. In the absence of an effect, it is necessary to proceed to surgical treatment [21].

Surgical treatment of generalized aggressive periodontitis is a set of measures aimed at preventing further tissue damage and bone loss in case of late diagnosis or inefficiency of therapeutic intervention. To minimize undesirable effects, an alternative laser operation can be used. With extensive loss of periodontal tissues, surgical treatment can lead to increased tooth mobility and deterioration of the clinical situation in general. Therefore, a careful assessment of the risk and benefit factors is necessary for a particular individual situation [22]. At present, reconstructive surgical treatment is based on the restoration of the anatomical form and functions of periodontitis. Bone replacement transplants, directed tissue regeneration (OTO) and biological modifiers (growth and differentiation factors, extracellular matrix proteins) are widely used.

To eliminate pathological foci, it is useful to perform patchwork operations on soft tissues. The repositioned flap (Widmann's modified flap) and the flap folding technique while maintaining the gingival papilla are the methods of choice in the dental practice. The combination of surgical treatment with systemic antibiotics leads to a greater reduction in the microbial load and the depth of the pocket.

The bone fractures that occur in a short time with aggressive periodontitis are a particular problem and require immediate action by a doctor. There are two methods with the most successful treatment results - bone transplantation and targeted tissue regeneration. The dental materials market offers a wide range of transplant materials, including autografts, alloimplants, xenoimplants and alloplastics. In progress in autotransplantation, material is transplanted from one anatomical area to another within the same organism. Autogenic transplants are considered a benchmark and have excellent regenerative properties, which have been scientifically proven in histological examination. Additional advantages are the elimination of the risk of the spread of the disease, the absence of an immune response and the reduction of the cost of surgery. The main limitations in the use of autografts in periodontal regeneration are the limited amount of bone tissue that can be moved, combined with a high risk of developing pathological processes in the donor site [23]. Nowadays, advances in tissue engineering and biomaterial technology represent a large number of treatment options for bone defects, which is why doctors usually use alloimplants, xenoimplants and alloplastic bone substitutes in their practice. A bone transplant between two genetically different organisms is called an alloimplantation. Bone alloimplants are unique in that they are usually removed together with the cellular component to reduce the risk of rejection. In addition, they are carefully treated to exclude any possibility of transmission of the disease. Clinical studies confirm the favorable outcome of treatment with these materials. With regard to the filling of intraosal defects, more than 50% of them completely dissolve [24]. The use of animal materials for the repair of human tissues is based on the use of bovine or coral material. These types of implants are called xenoimplants. The combination of bovine material with purified porcine collagen or synthetic polypeptide (PepGen P-15) stimulates regeneration and leads to the formation of a healthy periodontal ligament. Due to the low allergenicity and biodegradation of collagen, it has good compatibility with tissues. Collagen preparations reduce inflammatory reactions and shorten the wound healing time [25]. Synthetic bone substitutes include osteoconductive polymers in the form of blocks, pellets, cements or osteoinductive proteins. Proteins stimulate osteogenesis, cementogenesis and the growth of periodontal ligaments, and the osteoconductors are a passive matrix for the new bone. Hydroxyapatite, beta-tricalcium phosphate and bioactive glass are most often used as alloplastic materials [24].

The principle of directed tissue regeneration is based on the use of membranes that prevent the migration of the gingival epithelium and the growth of granulation tissue, as a result of which specific, poorly differentiated periodontal ligament cells can form a new connective tissue attachment. The preparation of the membrane is possible from biological preparations or from the patient's blood. One of the most commonly used is a membrane made of stretched polytetrafluoroethylene. Numerous experimental and clinical studies have proven the effectiveness of membrane technology in combination with the planting material. This method is being actively improved, the technology for the production of membranes is being improved, but today the guaranteed effectiveness can be found only with defects on 3 walls [26].

LITERATURE

1. Armitage G.C. Development of a classification system for periodontal diseases and conditions // Annals of Periodontology. 1999. Vol. 4(1). P. 1–6.
2. Cho C-M., You H-K., Jeong S-N. The clinical assessment of aggressive periodontitis patients // Journal of Periodontal & Implant Science. 2011. Vol. 41(3). P. 143-148.
3. Demmer R.T., Papapanou P.N. Epidemiologic patterns of chronic and aggressive periodontitis // Periodontology 2000. 2010. Vol. 53(1). P. 28–44.
4. Stabholz A., Soskolne W.A., Shapira L. Genetic and environmental risk factors for chronic periodontitis and aggressive periodontitis // Periodontology 2000. 2010. Vol. 53(1). P. 138–153.
5. De Carvalho F.M., Tinoco E.M.B., Govil M., Marazita M.L., Vieira A.R. Aggressive periodontitis is likely influenced by a few small effect genes // Journal of Clinical Periodontology. 2009. Vol. 36(6). P. 468–473.
6. Waerhaug J. Plaque control in the treatment of juvenile periodontitis // Journal of Clinical Periodontology.1977. Vol. 4(1). P. 29–40.
7. Guarnelli ME., Zangari F.., Manfrini R, Scapoli C., Trombelli L. Evaluation of additional amine fluoride/stannous fluoride-containing mouthrinse during supportive therapy in patients with generalized aggressive periodontitis: a randomized, crossover, double-blind, controlled trial // Journal of Clinical Periodontology. 2004. Vol. 31(9). P. 742–748.
8. Haber J., Wattles J., Crowley M., Mandell R., Joshipura K., Kent R..L. Evidence for cigarette smoking as a major risk factor for periodontitis // Journal of Periodontology. 1993. Vol. 64(1). P. 16–23.
9. Apatzidou D.A., Kinane D.F. Quadrant root planing versus same-day full-mouth root planing. I. Clinical findings // Journal of Clinical Periodontology. 2004. Vol. 31(2). P. 132–140.
10. Santos V.R., Lima J.A., De Mendonça A.C., Braz Maximo M.B., Faveri M., Duarte P.M. Effectiveness of full-mouth and partial-mouth scaling and root planing in treating chronic periodontitis in subjects with type 2 diabetes // J Periodontol. 2009 Aug. Vol. 80(8). P. 1237–1245.
11. Aimetti M., Romano F., Guzzi N., Carnevale G. One-stage full-mouth disinfection as a therapeutic approach for generalized aggressive periodontitis // Journal of Periodontology. 2011. Vol. 82(6). P. 845–853.
12. De Oliveira R.R., Schwartz-Filho H.O., Novaes A.B. et al. Antimicrobial photodynamic therapy in the non-surgical treatment of aggressive periodontitis: cytokine profile in gingival crevicular fluid, preliminary results // Journal of Periodontology. 2009. Vol. 80(1). P. 98–105.
13. Kamma J.J., Vasdekis V.G.S., Romanos G.E. The effect of diode laser (980 nm) treatment on aggressive periodontitis: evaluation of microbial and clinical parameters // Photomedicine and Laser Surgery. 2009. Vol. 27(1). P. 11–19.
14. Tomasi C., Schander K., Dahlen G., Wennström J.L. Short-term clinical and microbiologic effects of pocket debridement with an Er:YAG laser during periodontal maintenance // J Periodontol. 2006. Vol. 77. P. 111–118.
15. Takasaki A.A., Aoki A., Mizutani K. et al. Application of antimicrobial photodynamic therapy in periodontal and peri-implant diseases // Periodontology 2000. 2009. Vol. 51(1). P. 109–140.
16. Haffajee A.D., Socransky S.S., Gunsolley J.C. Systemic anti-infective periodontal therapy. A systematic review // Annals of Periodontology. 2003. Vol. 8(1). P. 115–181.

МЕТОДЫ ЛЕЧЕНИЯ ГЕНЕРАЛИЗОВАННОГО АГРЕССИВНОГО ПАРОДОНТИТА

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Аннотация*. Агрессивный пародонтит — это разновидность пародонтита, характеризующаяся молниеносным разрушением периодонтальной связки и потерей альвеолярной костной массы и возникающая, как правило, у молодых людей, реже у пожилых пациентов [1]. Согласно классификации заболеваний пародонта, принятой на заседании Президиума секции пародонтологии Стоматологической ассоциации России в 2001 году, это заболевание было выделено в самостоятельную группу заболеваний пародонта - агрессивные формы пародонтита. Однако стоматологи чаще обращаются к классификации стоматологических заболеваний МКБ-С-3 (ICD-C-3), основанной на МКБ-10 (ВОЗ, 1995), согласно которой агрессивному пародонтиту соответствует код к 05.4 "пародонтит". Нами проводились исследования популяции из 267 пациентов (165 мужчин (60,3%) и 102 женщин (39,7%)), которые составили 89 случаев (1,65%). Не было обнаружено существенных различий между процентным соотношением пациентов мужского и женского пола. Генерализованная форма была более распространена, чем локализованная, в соотношении 27:1.*

Ключевые слова: *болезни, излучение, лечение, диагностика.*