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SHOULD ANTIBIOTICS BE USED IN COMPLEX THERAPY OF CHRONIC INFLAMMATORY PERIODONTAL DISEASES

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Abstract:

The article is devoted to the topical issues of including antibiotics in complex treatment of patients with chronic inflammatory periodontal diseases.

The aim of the work is to give an extensive analysis of the problem of applicability and effectiveness of antibiotics in terms of etiology and pathogenesis of chronic inflammatory periodontal diseases.

The concept of etiology and pathogenesis of chronic generalized periodontitis as a multifactorial systemic disease resulting from imbalance between aggressive factors (periodontal pathogens) and body protection factors, oral cavity and periodontal complex, is discussed. On the basis of modern scientific data, the interaction of the human body and oral microflora in normal pathology has been analyzed. The problem of irrational use of antibiotics in Periodontology has been shown and the mechanisms of development of antibiotic resistance of periodontopathogenic microorganisms have been described.

The modern principles of antibiotic therapy and methods of using antibiotics in treatment of periodontics have been discussed. It is noted that identification of microflora and its sensitivity to antibiotics is necessary in determining periodontal pathogens found in the periodontal pockets.

Keywords: *inflammatory periodontal diseases, antibiotic therapy, antibiotic resistance.*

Introduction

Introduction: The diversity of a number of symptoms and characteristics of the course of inflammatory periodontal diseases, the presence of combined forms of pathology (comorbid conditions) in modern humans complicates the problem of clearly distinguishing these diseases. A necessary prerequisite for chronic periodontal inflammation is the impossibility of completing acute periodontal inflammation by regeneration, which is determined by the characteristics of the macroorganism's reaction, the anatomical and physiological characteristics of the periodontium and the specificity of pathogenic factors that have an adverse effect on it.

Unfortunately, the current stage of the study of inflammation is characterized by clearly insufficient knowledge about the nature of chronic inflammation, especially periodontal inflammation [1, 2].

With further progression of gum inflammation, the destruction of the periodontal junction, the formation of periodontal pockets, resorptive changes in the alveolar process (part) of the lower (upper) jaw occur, the disease acquires the typical clinical and morphological features of chronic periodontitis of varying severity.

The main difference between gingivitis and periodontitis, as some authors believe [3], is the topic, severity and severity of inflammation, the degree of involvement of various periodontal structures in it.

According to the authors of this article, chronic generalized periodontitis is, firstly, a multifactorial systemic (and not infectious in the classical sense) disease that occurs under the influence of the unfavorable cumulative effects of exogenous general and local factors (infectious agents of biofilm - according to modern views, biofilms are three-dimensional structures formed by bacteria on the surface of substrates, very resistant to antibiotics; anomalies of frenulum attachment; defects in fillings, prosthetics; anomalies in the position of teeth; malocclusion) and endogenous influences (diseases of the gastrointestinal tract, hormonal disorders, blood diseases), infectious-induced immune damage to the periodontal complex with a high probability of genetic predisposition; secondly, chronic generalized periodontitis is a disease resulting from an imbalance between aggressive factors (periodontopathogens) and protective factors of the macroorganism, mouth and periodontal complex, occurring with initial (initial) damage to the gums (gingivitis) and subsequent (and/or parallel to current) involvement in the pathological process of other periodontal structures (periodontitis); thirdly, chronic generalized periodontitis is a disease characterized by a progressive wave-like course (i.e., repeated periods of exacerbations and remissions) resulting in bone resorption in the alveolar process (part) of the lower (upper) jaw, destruction of the tooth retaining apparatus, formation of a periodontal pocket and ending (as a rule, without timely and adequate treatment) with tooth loss or removal, elimination of periodontal disease and undoubted dysfunction of the dental system and the body as a whole.

In the process of evolution over many millennia, as a result of natural selection in the selection of microorganisms, the endoecological system of functioning “macroorganism-endosymbiont bacteria” arose and became established. In its historical development, it went through (conditionally) several stages, the last of which began in the middle of the 20th century in the “era of antibiotics” and was accompanied by the partial destruction of endosymbiont microflora [4].

If we assume that both gingivitis and periodontitis in humans are caused by a microorganism that has invaded the human body, then following the postulates ("triad") of R. Koch, the following must take place: 1) the microbe that causes a particular disease must always be detected in the patient's body , but should not occur in healthy people or other diseases;

2) the microbe that causes the disease can be isolated from the patient's body in pure culture and cultivated outside it (on bacterial media); 3) the microbe that causes the disease, isolated in pure culture, when introduced into the body of a person susceptible to it, should cause a similar disease in him. And, therefore, eradication (destruction) of the microbe that causes the disease should lead to recovery of the patient.

In this regard, it is appropriate to recall the statement of I.V. Davydovsky (1962): "A cause that does not act is not a cause at all" [5].

In the light of modern knowledge, chronic generalized periodontitis is considered as a multifactorial disease, thereby emphasizing the absence of a specific etiological factor.

"Tooth" plaque is a necessary component for the development of periodontal diseases, but its absolute

Its role as a root cause of periodontal inflammation may be questioned [6]. In the pathogenesis of periodontitis, the immune system decides a lot [7, 8], and the active participation of periodontopathogenic microflora in the inflammatory process [9] and microcirculation disorders in the periodontium have also been proven.

The most important feature of the oral cavity is that the processes occurring in it are carried out in the presence of microbes. It is believed that inflammatory periodontal diseases occur with the participation of representatives of the body's permanent microflora, which are weakly pathogenic or complete saprophytes [10].

The presence of over 500 species of microorganisms in the oral cavity, the possibility of their combined action, and the variability of the composition of saprophytic microflora present significant difficulties in assessing the role of bacteria as etiological and pathogenetic factors in the development and further progression of inflammatory-destructive periodontal diseases [11].

Many provisions of the "infectious origin" of inflammatory periodontal diseases are unproven and are so far only taken "on faith".

There are still certain difficulties in providing medical care to patients with periodontal pathology.

If just a few years ago, the elimination of biofilm (eradication, or reduction in the number of periodontal pathogenic bacteria) and periodontal pockets were at the forefront of methods of treating developed (and actively ongoing) periodontitis, now the therapeutic arsenal has been replenished with many other approaches and possibilities for combating periodontitis: replantation of bone tissue and artificial materials; technique of directed tissue regeneration; use of matrix proteins and growth factors; photodynamic therapy; suppression of biofilm (microbial initiation of inflammation), etc. [12-15].

It should be noted that no standards have yet been proposed for drug therapy for inflammatory periodontal diseases, including the use of antibiotics, although the arsenal of antibacterial therapy is quite wide (gels, rinses, solutions, emulsions, diplene films, balms, gum dressings, etc.) [16].

Researchers and clinicians recognize the obvious fact that irrational antibiotic therapy (prescription "just in case", inappropriate dose, drug or course duration, unjustified combinations of antibiotics, etc.) is a common problem [17].

As a result, the threat of development of resistance of microorganisms to standardly used antibiotics increases, which in turn necessitates the use of new and, as a rule, more expensive drugs with often an increased level of side effects.

It is noted [18] that in the domestic literature there is no shortage of publications on this problem, but, unfortunately, they are “scattered” across a large number of “scientific” journals, “hidden” between articles on other topics and advertising materials. And outside Russia the picture is similar. Thus, according to a study by K. Fleming-Dutra et al., in the United States, when providing outpatient care, about 30% of antibiotic prescriptions are unnecessary.

Antibiotic sensitivity and resistance.

Antibiotic resistance of microorganisms is one of the most pressing problems of modern medicine. Drug-resistant bacteria are rapidly spreading across the planet. According to the Ministry of Health of the Russian Federation, about 16% of Russians today have antibiotic resistance to various drugs.

At the same time, 46% of the Russian population are convinced that antibiotics “kill” viruses in the same way as bacteria [19]. In Russia, the issue of preventing the development of bacterial resistance to existing antibacterial drugs is especially acute due to the over-the-counter supply of antibiotics and the lack of sufficient public awareness of this problem [12].

The authors of this study presented data on almost 20 types of medical errors when using, for example, beta-lactam antibiotics. More than half of these errors are a consequence of various violations of the dosage regimen, prescriptions in the presence of contraindications, and violations of the frequency of use.

When treating patients with inflammatory periodontal diseases, the prescription of antibacterial therapy is due to the fact that bacterial pathogens are located in bone and soft tissues, as well as in biofilm (superficial and subgingival), and not only on the surface of the oral mucosa or in the exudate of the periodontal pocket [20].

The main problem is that the modern strategy for combating bacterial infections is focused on the destruction of individual microbes, not biofilms, which have an incredible ability not only to evade the body's protective factors, but sometimes stop responding to antibiotics used.

Such super-endurance is given to biofilms by the matrix, the presence of DNA in which can be destroyed, for example, using the enzyme DNase [21]. To monitor the effectiveness of treatment for patients with periodontitis, molecular biological methods for microbiological examination of the contents of periodontal pockets are recommended [22].

The arsenal of antibacterial agents is huge. Systemic administration of antimicrobial agents in conventional doses does not allow them to reach a concentration sufficient to destroy bacteria protected by biofilm in the subgingival space and in periodontal pockets.

In inflammatory periodontal diseases, local antimicrobial therapy is attempted by irrigating the pockets or introducing ointments, gels containing antibiotics, as well as long-acting agents designed to create a high concentration over a certain period of time [23].

It is not always taken into account that for effective antibacterial therapy it is recommended to provide access to periodontal pathogenic microorganisms, including those in a deeply structured biofilm, i.e. outside the tissues supplied with blood and effectively reduce their concentration [24].

Principles of antibiotic therapy It is recommended to strictly adhere to ten principles of strategy and tactics for the rational use of antimicrobial agents in outpatient settings.

1. Antimicrobial drugs (AMPs) should be prescribed only if there are reasonable indications - the presence of a documented or suspected bacterial infection (except for limited cases of antibiotic prophylaxis).
2. The choice of the optimal regimen of antibacterial therapy (ABT) should be made taking into account the pharmacokinetics and pharmacodynamics of the antibiotic and implies the appointment of an adequate antibiotic in an adequate dose with a planned adequate duration of therapy.
3. When choosing antimicrobial agents, it is necessary to know the regional situation with antibiotic resistance of the most relevant pathogens and take into account the patient's risk of infection with this resistant pathogen.
4. Avoid prescribing AMPs of low quality and with unproven effectiveness.
5. Avoid unjustified prophylactic prescription of antibacterial, antifungal and antiviral agents.
6. The initial assessment of the effectiveness of AMT should be carried out within 48-72 hours after the start of treatment.
7. Explain to patients the harm of non-compliance with the prescribed ABT regimen and the danger of self-medication with antibiotics.
8. Promote patients' compliance with the prescribed regimen for the use of antimicrobial agents (drug, daily dose, frequency of administration, duration of use).
9. Use the capabilities of the microbiological laboratory in practical work and actively implement express methods for the etiological diagnosis of infections.
10. Use expert practice recommendations based on evidence-based medicine as a guide.

In dental practice, about 20 different groups of antibacterial drugs are used (penicillins, cephalosporins, macrolides, tetracyclines, lincomycins, nitroimidazoles, quinolones), each of which has a well-studied mechanism of action, pharmacological properties and antimicrobial spectrum and has different routes of administration (oral, intramuscular, intravenous, local) and frequency (single, once/several times a day) of administration. Local delivery of antimicrobials requires the same caution as systemic administration.

Determining the microbiological spectrum of the contents of periodontal pockets allows you to select the most effective remedy. Systemic antibacterial therapy is recommended [26] only when odontogenic infection spreads beyond the periodontal period (under the periosteum, into bones, soft tissues of the face and neck), in the presence of elevated body temperature, regional lymphadenitis, intoxication and insufficiency/inadequacy of surgical intervention (for example, opening a periodontal abscess during exacerbation of periodontitis). In dental practice, in the treatment of such "infectious diseases" as periodontitis, after diagnosis (history, clinical examination, comorbid conditions, x-ray graphical examination) determines the indications for systemic antimicrobial therapy (local or systemic infection) [27]. At the same time, restorative and sanitation (for example, regarding caries and its complications) measures are carried out.

In the literature available to us, we did not find data proving that the use of antibiotics simultaneously with periodontal surgical interventions (curettage, flap operations, etc.) improves the outcome of the treatment. However, a special infection control regimen, usually including antibiotics, is recommended for implants, resorbable and non-resorbable membranes [28].

To prevent infections associated with bacteremia in high-risk patients (infective endocarditis, prosthetic heart valves, heart defects, hemodialysis, immune disorders), different antibiotic regimens have been proposed [29].

Conclusion

A mandatory step in identifying periodontopathogens found in periodontal pockets of patients is the identification of microflora and its sensitivity to antibiotics. This largely depends on the interaction between the dentist (who strictly follows the rules for collecting microbiological material from the periodontal pocket) and the laboratory [30].

Studies show that after discontinuation of antibacterial therapy, its positive effect ends [31, 32]. In addition, not all clinicians take into account the fact that the prescription of antibacterial therapy, even taking into account the sensitivity of the microflora, does not bring the expected effect.

Apparently, this is due to the peculiarities of the connection between the immune, nervous, hematopoietic and other systems that regulate the protective reactions of the body and periodontal tissues during inflammation of the latter [33, 34]. Recent publications [35] emphasize that any drug therapy carries a potential risk of adverse drug reactions, being an iatrogenic event with all the ensuing negative consequences (clinical, economic, legal, etc.). Antibacterial drugs occupy a leading position among drugs that cause unwanted reactions, including in our country. The search for more effective pharmaceutical substances and drugs based on them continues [24, 36].

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