

# Etiotropic therapy of aggressive periodontitis: Contemporary antibiotic regimens and their effectiveness

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**Abstract. Introduction.** Aggressive periodontitis is a rapidly progressing periodontal disease characterized by swift destruction of the periodontal complex tissues with minimal clinical signs of inflammation. Despite the paucity of symptoms, the disease is accompanied by significant bone loss, which often leads to diagnostic challenges. **Aim.** To conduct a systematic analysis of contemporary antibiotic regimens for the treatment of aggressive periodontitis, evaluating their efficacy, pharmacological characteristics, and clinical advantages based on global literature data. **Materials and Methods.** A comprehensive search and critical analysis of scientific publications indexed in Scopus, Web of Science, and PubMed was performed. The study included publications focused on etiotropic therapy for patients with various forms of aggressive periodontitis, published between 2015 and 2025, with a minimum follow-up duration of six months. Experimental studies and case reports were excluded. In total, 55 publications were analyzed. **Results and Discussion.** It was found that in clinical practice, the most frequently used antibiotics for the treatment of aggressive periodontitis include macrolides (500 mg once daily for 3 days), fourth-generation fluoroquinolones (400 mg once daily for 7 days), as well as combination regimens comprising  $\beta$ -lactam antibiotics with nitroimidazoles, with dosage and course duration varying across studies. The preference for macrolides and fluoroquinolones is primarily based on their favorable pharmacokinetic properties, particularly their ability to penetrate deeply into inflamed tissues and accumulate within innate immune effector cells. Combination of amoxicillin and metronidazole demonstrates a pronounced synergistic effect, including suppression of matrix metalloproteinase production and broad-spectrum antibacterial coverage encompassing both aerobic and anaerobic pathogens. Interregional variations observed in microbial composition underscore the necessity of personalized etiotropic therapy tailored to local microbial profiles and pathogen susceptibility. **Conclusions.** Despite extensive research into the etiotropic management of aggressive periodontitis and endorsement of the antibacterial approach by leading professional associations, a standardized treatment protocol has not yet been established. The synthesized evidence emphasizes the importance of individualized antibiotic therapy, taking into account the microbial spectrum, clinical course, and pathogen sensitivity. Future research prospects involve enlarging sample sizes and conducting comparative analyses of different antibiotic regimens to enhance the efficacy of aggressive periodontitis management.

**Keywords:** aggressive periodontitis, antibiotic therapy, etiotropic treatment, microbial spectrum, macrolides, fluoroquinolones, clinical efficacy

**For citation:** Gaybullaev, E.A. Etiotropic therapy of aggressive periodontitis: contemporary antibiotic regimens and their effectiveness. The Bulletin of Contemporary Clinical Medicine. 2026, 19 (1), 118-122. DOI: 10.20969/VSKM.2026.19(1).118-122.

# Этиотропная терапия агрессивного пародонтита: современные схемы антибактериальной терапии и их эффективность

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**Реферат. Введение.** Агрессивный пародонтит представляет собой быстро прогрессирующее пародонтологическое заболевание, характеризующееся стремительной деструкцией тканей пародонтального комплекса при минимальных клинических проявлениях воспаления. Несмотря на скудную симптоматику, заболевание сопровождается значительной потерей костной ткани, что нередко приводит к диагностическим ошибкам. **Цель.** Провести систематический анализ современных схем антибиотикотерапии агрессивного пародонтита, оценить их эффективность, фармакологические особенности и клинические преимущества на основании данных мировой литературы. **Материалы и методы.** Выполнен углублённый поиск и критический анализ научных публикаций, включённых в базы данных Scopus, Web of Science и PubMed. В исследование вошли работы, посвящённые этиотропной терапии пациентов с различными формами агрессивного пародонтита, опубликованные в 2015-2025 гг., с длительностью наблюдения не менее 6 месяцев. Исключены экспериментальные исследования и клинические случаи. Всего проанализировано 55 публикаций. **Результаты и их обсуждение.** Установлено, что в клинической практике лечения агрессивного пародонтита наибольшее распространение получили антибиотики ряда макролидов (в стандартной дозировке 500 мг 1 раз в сутки на протяжении 3 дней), фторхинолоны IV поколения (400 мг 1 раз в сутки в течение 7 дней), а также комбинированные схемы, включающие  $\beta$ -лактамы антибактериальные препараты в сочетании с нитроимидазолами, варьирующиеся по дозировке и длительности курса. Преимущественный выбор макролидов и фторхинолонов обусловлен их благоприятными фармакокинетическими характеристиками, в частности способностью к глубокому проникновению в воспалённые ткани и аккумуляции в клетках-эффекторах врождённого иммунитета. Комбинация амоксициллина и метронидазола демонстрирует выраженный синергетический эффект, сопровождающийся подавлением продукции матриксных металлопротеиназ и обеспечением широкого антибактериального покрытия, включающего как аэробные, так и

анаэробные патогены. Установленные межрегиональные различия в структуре микробиоты подчёркивают необходимость персонализированного выбора этиотропной терапии, ориентированного на локальные особенности микробного спектра и чувствительности возбудителей. **Выводы.** Несмотря на активное изучение этиотропной терапии агрессивного пародонтита и поддержку антибактериального подхода ведущими профессиональными ассоциациями, единый протокол лечения отсутствует. Систематизированные данные подчёркивают необходимость индивидуального выбора антибактериальной терапии с учётом микробного спектра, клинического течения и чувствительности возбудителей. Перспективы дальнейших исследований связаны с расширением выборок и сравнительным анализом различных схем антибиотикотерапии для повышения эффективности лечения агрессивного пародонтита.

**Ключевые слова:** агрессивный пародонтит, антибактериальная терапия, этиотропное лечение, микробный спектр, макролиды, фторхинолоны, клиническая эффективность

**Для цитирования:** Гайбуллаев Э.А. Этиотропная терапия агрессивного пародонтита: современные схемы антибактериальной терапии и их эффективность // Вестник современной клинической медицины. – 2026. – Т. 19, вып. 1. – С. 118–122. DOI: 10.20969/VSKM.2026.19(1).118-122.

**I ntroduction.** Aggressive periodontitis in the modern classification refers to acute periodontal diseases, with rapid or, in some cases, lightning-fast development of the pathological process in the tissues of the periodontal complex, significant loss of a large volume of bone tissue with minimal clinical manifestations and subjective complaints from the patient. Quite often, during a clinical examination, there are no significant inflammatory changes in the oral cavity, which very often leads to diagnostic errors due to the visible weak inflammatory response of the tissue. A large number of publications have appeared in the modern literature on the role of immune mechanism imbalance both in the oral cavity and in the body as a whole, while a large number of microorganisms are not always isolated from the periodontal pockets' characteristic of aggressive periodontitis. All of the above is not proportional to the severity of the developing destruction of the periodontal complex tissues [1, 2, 3]. Initially, aggressive periodontitis as an independent nosologically unit was classified in the 1990s by the American Academy of Periodontology [4], according to which periodontal pathology was divided into "aggressive" and "chronic" forms based on the course of the pathological process. Around the same time, this classification was supplemented with an assessment of the prevalence of the pathological process, which led to the division into "generalized" and "local" forms of periodontal pathology. An analysis of the scientific literature has led to the conclusion that most scientific research focuses on the etiology and pathogenesis of the chronic form of periodontal inflammation, as it is the most common in the population. It is known that aggressive periodontitis is quite rare, with a prevalence of no more than 1-3% in developed countries in Europe and the USA, while in Central Asia, Russia, and African countries, the prevalence increases to 5-7% of the population [4,5]. From the point of view of a practicing dentist, aggressive periodontitis, as a rule, does not have a pronounced inflammatory picture in the periodontal region. Patients may have a small amount of dental plaque and biofilms, but when performing an overview X-ray of the periodontal region, pronounced bone tissue destruction is revealed. In the literature, many researchers explain this phenomenon by the formation of an imbalance in the local immunity of the periodontal region and its nonspecific defense factors (the state of effector cells) [7,8]. The clinical course of aggressive periodontitis is characterized by recurrent

episodes with a frequency of once every 3-4 months; in unfavorable, rapidly progressive cases, there may be no remissions [9]. At the beginning of the 21st century, a sufficient number of scientific publications appeared on the role of hereditary factors as the leading etiological factor in the development of aggressive periodontitis. However, today this trend towards identifying the leading etiological factor and considering aggressive periodontitis from the perspective of monocausalism has practically disappeared [3, 5]. Most authors agree on the polyetiological nature of aggressive periodontitis development with the mandatory participation of an external etiological factor in the form of pathogenic microorganisms. There are a sufficient number of studies in the literature describing the main types of microorganisms that have high invasive and toxic properties on periodontal tissues. To date, more than 400 types of microorganisms have been identified in the periodontal region that are capable of disrupting the protective barriers of the periodontal region to one degree or another. However, scientists agree that of this species diversity, only three have the greatest ability to invade the periodontal mucosa, namely *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis* and *Tannerella forsythia* [10, 11]. At the same time, a large number of studies have been devoted to the study of the pathogenic properties of *Aggregatibacter actinomycetemcomitans* as the main causative agent of periodontal disease [12, 13]. In recent years, studies have been published on the pathogenicity and tropism to the periodontal disease mucosa of *Prevotella intermedia* and *Treponema denticola* [14]. In view of the above, a systematic approach to the choice of antibiotic therapy is relevant for the treatment of aggressive periodontitis, since the physician must take into account that this form of pathology develops rapidly against the background of an erased course of the pathological process [15, 16].

The literature already contains descriptions of studies devoted to the selection of antibiotic therapy, its dosage, regimen, and frequency of administration, as well as the assessment of local and general application [17]. Despite the caution surrounding the prescription of antibacterial therapy in European countries and the USA, both the European Federation of Periodontology and the American Academy of Periodontology support the additional use of antibacterial therapy in the treatment of aggressive periodontitis [18, 19]. Despite the annual consensus reports and protocols, there is no

unified protocol and regimen for prescribing antibiotic therapy for aggressive periodontitis [20, 21].

This served **as the aim of the present study** — to analyze the most effective methods and dosing regimens of antibiotic therapy in patients with aggressive periodontitis based on the literature.

#### **Materials and Methods.**

A search and in-depth analysis of current scientific literature data on improving the effectiveness of aggressive periodontitis treatment and developing algorithms and treatment regimens for aggressive periodontitis antibiotic therapy was performed.

Inclusion criteria: Analysis of scientific papers devoted to the problem of etiotropic treatment of patients with various forms of aggressive periodontitis published from 2015 to 2025 with a study period of at least 6 months. All papers are published in the Scopus, Web of Science, and PubMed scientific citation databases.

Exclusion criteria: experimental studies and descriptions of clinical cases.

#### **Results and Discussion.**

As a result of an in-depth search using an artificial intelligence system, 55 publications were included in the study. Most of the scientific papers were published by scientists from Europe and the USA [20, 22, 23], Turkey [24, 25], and others. The minimum study duration was 6 months, and the maximum was 4 years [26].

Scientific studies have examined the mechanisms of action and clinical efficacy of various antibacterial agents and their combinations in the management of aggressive periodontitis. Fourth-generation fluoroquinolones were administered at 400 mg once daily for a 7-day course, while macrolides were prescribed at 500 mg once daily for a 3-day course [27]. In the case of combination therapy involving semisynthetic penicillins and 5-nitroimidazole derivatives, diverse dosing regimens were applied: both agents at 500 mg three times daily for 7 days, or the penicillin derivative at 500 mg combined with the nitroimidazole at 250 mg three times daily for 7–10 days, or the penicillin at 875 mg with the nitroimidazole at 500 mg twice daily for 10 days, and alternatively, the penicillin at 375 mg combined with the nitroimidazole at 250 mg three times daily for 7 days [28].

Each of the drug dosing regimens was proposed by the authors as the most effective, with clear advantages over standard dosing regimens [29]. The pharmacokinetics and pharmacodynamics of antibacterial drugs from the macrolide and fluoroquinolone groups, their ability to accumulate in biological fluids, including mixed saliva, and selectively inhibit the activity of the main pathogenic microorganisms (*Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, and *Tannerella forsythia*) [30].

The authors evaluate the preference for macrolides based on their ability to accumulate in the primary inflammatory focus and penetrate segmented neutrophils and epithelial cells [31]. In addition, the main factor in choosing macrolides and fourth-generation fluoroquinolones is the convenient dosage regimen and duration of treatment: 400 mg once daily or 500 mg once daily, respectively, with a total duration of treatment from 3 to 7 days, which allows avoiding the main side effects of antibiotic therapy [32, 33]. The pharmacokinetics of

macrolides are based on a long half-life of up to 65 hours, which allows the drug to remain in the gingival mucosa at therapeutic concentrations for up to 7 days after the completion of the course of antibiotic therapy. Delivery to the tissue is carried out through a system of mononuclear cells (phagocytes, neutrophils) that are directed to the site of inflammation by chemotaxis. There are a number of studies confirming that the more severely the tissue is damaged by inflammation, the more effector cells are directed there, and therefore, the higher the local concentration of the drug [34]. The active substance penetrates the phagocytic cells and fibroblasts and accumulates in them, increasing its intracellular concentration by 10 to 100 times compared to the extracellular concentration. During active killing, the phagocyte is destroyed, releasing a significant amount of the drug [35, 36].

The prescription of a combination of semisynthetic penicillins and 5-nitroimidazole derivatives (amoxicillin + metronidazole) by doctors in the etiopathogenetic treatment of aggressive periodontitis is quite widespread due to their synergistic antibacterial action, covering a wide range of pathogens [37,38,39]. According to a number of authors, drugs from the group of semisynthetic penicillins (amoxicillin) disrupt the synthesis of membrane protein in bacterial cells, i.e., disrupt the synthesis of the cell wall, while 5-nitroimidazole derivatives (metronidazole) damage the DNA molecule of the bacterial cell and have a bactericidal effect. Thus, the combination of these groups of drugs increases the number of destroyed microorganisms, ensuring complete coverage of  $\alpha$ - and anaerobic microorganisms associated with aggressive periodontitis without causing a high risk of antibiotic resistance [40, 41, 42].

In combination with clavulanic acid, amoxicillin (Amoxiclav, Augmentin) can also be used to suppress  $\beta$ -lactamase-producing bacteria [43]. This enzyme is secreted by microorganisms to destroy penicillins, therefore the use of clavulanic acid will neutralize it and restore sensitivity to amoxicillin, thereby increasing the effectiveness of therapy [44].

The advantage of using a combination of amoxicillin and metronidazole is its ability to reduce the expression of matrix metalloproteinases. It is known that the main groups of anaerobic pathogens associated with the development of aggressive periodontitis stimulate the production of major pro-inflammatory cytokines, which at high concentrations are capable of activating matrix metalloproteinases in gingival tissues [45].

The combination of these drugs with mechanical cleaning (scaling and root planing) is more effective than drug monotherapy, reducing pocket depth, reducing gingival bleeding, and restoring attachment [46].

Recently, scientific data has emerged indicating the peculiarities of the microbial content of periodontal pockets depending on the patient's region of residence and race [47]. Therefore, using the same generally accepted etiotropic treatment regimens is not always justified and does not produce a pronounced clinical effect. Therefore, in addition to the mandatory determination of the microbial profile, it is necessary to determine sensitivity/resistance to the drug and evaluate the tolerability of the dosage regimen [48, 49].

To determine the effectiveness of etiotropic therapy with antibiotics at a given time, there is currently a scale for its assessment based on the following key factors. The main ones are the form of the pathological process with localization of the focus, tartar or soft dental plaque deposition, depth of periodontal pocket, degree of bleeding, drug dosage regimen, drug side effects, and microbial resistance [50].

For an in-depth analysis of the effectiveness of etiotropic therapy in the pathogenesis of aggressive periodontitis, even larger sample sizes and studies are needed, providing statistical evaluation and comparative characteristics of various regimens for the use of antibacterial drugs in aggressive periodontitis.

**Conclusions.** To sum up the above, it can be concluded that despite the described mechanisms of action of the main groups of antibacterial drugs, interest in antibacterial therapy for aggressive periodontitis remains quite high. In the analyzed literature, most authors emphasize not only the study of drug combinations, but also the dosage regimen and selection of the most effective dose with minimal side effects, as well as the determination of sensitivity to antibacterial drugs in case of ineffective therapy.

**Transparency of the study.** *The study did not have sponsorship. The author is solely responsible for the provision of the final version of the manuscript for publication.*

**Прозрачность исследования.** *Исследование не имело спонсорской поддержки. Автор несет полную ответственность за предоставление окончательной версии рукописи в печать.*

**Declaration of financial or other relationships.** *The author developed the study concept and design and also wrote the manuscript. The author did not receive any honorarium for the study.*

**Декларация о финансовых и других взаимоотношениях.** *Автор разработал концепцию и дизайн исследования, а также написал рукопись. Автор не получал гонорар за исследование.*

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