Should Antibiotics Be Prescribed to Treat Chronic Periodontitis?

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KEYWORDS

• Antimicrobials-systemic • Periodontitis microbiology • Oral biofilm
• Periodontitis therapy • Scaling and root planing • Clinical trials

KEY POINTS

- Although chronic periodontitis often responds to mechanical debridement alone, patients with progressive attachment loss, invasive subgingival pathogens, or multiple deep pockets may benefit from combining systemic antibiotics with mechanical therapy.
- Bacteria in subgingival biofilm are resistant to antibiotics. Antibiotics should only be prescribed after biofilm has been mechanically disrupted, not as the sole approach to treatment.
- Meta-analyses suggest that metronidazole (in combination with amoxicillin or alone) or azithromycin produce statistically significant adjunctive benefits in combination with mechanical therapy.
- When used to treat chronic periodontitis, the combination of mechanical therapy and antibiotics yields its greatest benefit at sites with deep initial probing depths.
- Systemic antibiotics have the potential to produce adverse reactions that must be considered in balance with their expected benefits.

INTRODUCTION

Periodontitis is a chronic inflammatory disease that leads to destruction of the supporting tissues of teeth and, if left untreated, tooth loss. Severe periodontitis was the world's sixth-most prevalent condition in 2010; its age-standardized prevalence

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between 1990 and 2010 among all countries was 11.2%.[1] Consistent with this estimate, a study based on data from the 2009 and 2010 National Health and Nutrition Examination Survey cycle reported prevalence rates of 8.7%, 30.0%, and 8.5% for mild, moderate, and severe periodontitis, respectively, in the United States.[2]

Studies from the past 3 decades have revealed that only a small subset of microorganisms from among the hundreds of species found in the oral cavity is highly associated with periodontitis.[3] Although specific biofilm-producing bacterial pathogens and other cooperative species are required, bacteria alone are not sufficient to induce periodontitis. The host immune-inflammatory response is a determinant of susceptibility to periodontitis and is responsible for most of the periodontal tissue destruction.[4] During persistent bacterial infection and prolonged homeostatic imbalance, cytokines and enzymes released by host leukocytes mediate destruction of periodontal connective tissue and bone. Systemic diseases (eg, diabetes), immune dysfunction, and environmental factors (eg, smoking) can also contribute to disruption of the homeostatic balance.[5] The goal of periodontal therapy is to preserve the natural dentition in stability, comfort, and function by eliminating pathologic biofilm and resolving inflammation.

Microbial complexes in subgingival biofilm have been recently characterized using molecular techniques. Individual species in these complexes have been assigned using a color-coded system that reflects community ordination and cluster analysis.[6] The red complex, consisting of *Tannerella forsythia*, *Porphyromonas gingivalis*, and *Treponema denticola*, is strongly associated with severe chronic periodontitis. The orange complex, which includes *Prevotella intermedia*, *Fusobacterium nucleatum*, *Campylobacter rectus*, and *Peptostreptococcus micros*, is closely associated with the red complex. The green complex includes *Aggregatibacter actinomycetemcomitans*, which has a strong association with aggressive periodontitis and a less frequent association with chronic periodontitis.[7] *Porphyromonas gingivalis*, *A actinomycetemcomitans* and other pathogens possess virulence factors that can overcome the host response and damage periodontal tissues.[8,9]

Porphyromonas gingivalis, *A actinomycetemcomitans*, and *Prevotella intermedia* are capable of invading the epithelium of periodontal pockets, which protects them from elimination by the host response, making them exceptionally difficult to eliminate by conventional periodontal scaling and root planing (SRP). Persistent infections by these bacteria are frequently associated with progressive chronic periodontitis.[10] Another limitation of SRP is that it is not effective in removing bacteria from deep pockets, furcations, dentinal tubules, and other subgingival sites where access is poor. The difficulties associated with eliminating bacteria that have colonized the soft tissue wall of the pocket and other inaccessible areas provide a rationale for incorporating systemic antibiotics into the treatment of periodontitis.

A broad range of systemic antibiotics has been used to treat chronic periodontitis. The pharmacokinetic and antimicrobial properties of the agents used most commonly are presented in **Table 1** and information on dosage is detailed in **Table 2**. In general, amoxicillin, metronidazole, azithromycin, tetracycline, and doxycycline are capable of attaining levels that can effectively inhibit periodontal pathogens when they are growing as single (planktonic) cells in a periodontal pocket or the soft tissue wall of a pocket. The exception is metronidazole, which exhibits relatively poor activity against *A actinomycetemcomitans* at typical in vivo concentrations. However, it is important to remember that subgingival bacteria live in a biofilm, not as single cells. Bacteria growing in a biofilm are substantially more difficult to inhibit with antibiotics. For this reason, antibiotics should only be used to treat periodontitis in patients who have already had their subgingival biofilm disrupted by SRP.