Review Article

Clindamycin: An Unsung Hero in the management of periodontitis.

Maria Subash Aaron Muthuraj[1], Mary Kural Ayeni MAJ[2]

From, [1]Reader, Department of Periodontics, RVS dental college and Hospital, Sulur, Coimbatore - 641402, Tamilnadu, India, [2]Consultant, Elan Dental Care, Coimbatore-641006, Tamilnadu, India

ABSTRACT

Periodontitis is a polymicrobial multifactorial disease with dental plaque playing a key role in its initiation and progression. Most cases with periodontitis can be managed with nonsurgical therapy alone or followed by surgical periodontal therapy. But in some cases, after traditional periodontal therapy, there is a greater chance for recolonization of tissue-invasive microbes such as gram-negative anaerobic rods and cocci. In such cases, systemic antimicrobials can serve as an adjuvant to conventional periodontal treatment. Drugs like amoxicillin, tetracycline, azithromycin, doxycycline, moxifloxacin, etc., are usually given in such cases. Clindamycin, belonging to the lincosamide class of drugs, has remarkable potential in the management of periodontitis, particularly in cases of refractory periodontitis, acting on anaerobic bacteria. It can also be used in patients with penicillin allergies and in patients who are not responding to tetracycline therapy. Despite its serious side effects, like pseudomembranous colitis, its efficiency in refractory periodontitis is remarkable. It is one of the few antimicrobials that has both anti-microbial and immunomodulatory activity. The potential for clindamycin as a Local drug delivery in the management of periodontitis also been explored. Even though it has many positive attributes, it is still an underdog in periodontal treatment due to some notable but preventable side effects. This review considers the potential of clindamycin in the management of refractory periodontitis, along with its notable positive effects, such as immunomodulatory and bone-binding properties.

Key words: Clindamycin, Periodontitis, Chitosan.

Clindamycin is a commonly used lincosamide-class antibiotic, working by blocking microbial ribosomes. Normally, it is used to treat anaerobic bacterial infections. It can be used to treat diseases caused by protozoa, e.g., malaria. It is a common topical treatment for acne and can be useful against some methicillin-resistant Staphylococcus aureus (MRSA) infections. It is derived semi-synthetically from lincomycin, a natural antibiotic produced by the Actinobacterium Streptomyces lincolnensis. It is obtained by 7(S)-chloro-substitution of the 7(R)-hydroxyl group of lincomycin. The synthesis of clindamycin was first announced by BJ Magerlein, RD Birkenmeyer, and F Kagan at the fifth Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC) in 1966 [1].

It is specifically more active against gram-negative anaerobes associated with the periodontal microbial flora, making it very effective in the management of refractory periodontitis. A serious gastrointestinal disorder associated with its use is Pseudomembranous colitis [1]. Even though it is a preventable side effect, there is a lacuna in the narrative reviews written on clindamycin and its role in the management of periodontitis [2].

Access this article online

Received – 19th February 2026

Initial Review – 28th February 2026

Accepted – 2nd March 2026

Quick response code



In this narrative review, the mechanism of action, pharmacokinetics, bacterial spectrum, the role of clindamycin in the management of refractory periodontal diseases, its additional immunomodulatory property, and its possibility as a local drug delivery (LDD) agent are discussed.

2. FEATURES OF CLINDAMYCIN

2.1 Mechanism of action

Clindamycin exerts its bacteriostatic effect by inhibiting the synthesis of microbial proteins through its action on the 50S subunit of the bacterial ribosome [3].

2.2 Bacterial Spectrum

It is a broad-spectrum antimicrobial drug highly active against most gram-positive aerobic bacteria and a wide array of anaerobic bacteria, particularly against the bacteria producing beta-lactamase [4]. Eikenella corrodens is intrinsically resistant to the drug. It is not indicated in juvenile periodontitis, due to the in vitro resistance encountered with Aggregatibacter Actinomycetemcomitans [1].

2.3 Pharmacokinetics

Clindamycin is a well-absorbed oral drug, and it can penetrate all body fluids except the cerebrospinal fluid (CSF).

Correspondence to: Dr. Maria Subash Aaron Muthuraj, Department of Periodontics, RVS dental college and Hospital, Sulur, Coimbatore - 641402, Tamilnadu, India.

Email: subash.muthuraj@gmail.com

Clindamycin maintains concentrations more than the minimum inhibitory concentrations for most bacteria associated with adult periodontitis in the gingival crevicular fluid (GCF) [1].

In an in-vitro study, Clay B. Walker et al (1981) determined the susceptibility of periodontopathic bacteria to clindamycin and measured the GCF levels of this drug with a modified agar diffusion assay. Except for Eikenella corrodens and Aggregatibacter actinomycetemcomitans, almost all bacteria were inhibited by a concentration of 1.0 µg of clindamycin per ml or less. The peak concentrations achieved by the drug were approximately the same in the GCF (2.0 ± 0.3 µg /ml) and in the blood (1.9 ± 0.3 µg /ml). However, the GCF clindamycin levels were 1.0 µg/ml for up to 6 hours, whereas the drug concentration in blood dropped below 1.0 µg/ml within 2 hours after administration. On considering its minimal inhibitory concentrations (MIC), GCF levels of ≥ 1.0 µg /ml inhibit most bacteria associated with diseased periodontal sites [5].

2.4 Susceptibility of Bacterial Microflora to Clindamycin:

Clay Walker et al. (1981) reported susceptibility of all periodontal disease microflora to Clindamycin except Eikenella corrodens and Aggregatibacter actinomycetemcomitans [5]. Thomas E. Rams et al (2014) conducted a study to determine the in vitro susceptibility of fresh Streptococcus constellatus and Streptococcus intermedius clinical isolates from human periodontitis lesions to amoxicillin, azithromycin, clindamycin, ciprofloxacin, and doxycycline.

The strains were subjected to antibiotic gradient strip susceptibility testing with amoxicillin, azithromycin, clindamycin, ciprofloxacin, and doxycycline on blood-supplemented Mueller-Hinton agar and to the inhibitory effects of metronidazole at 16 mg/L in an enriched Brucella blood agar dilution assay. The results had shown that Clindamycin was the most active antibiotic against Streptococcus constellatus, and amoxicillin was the most active against Streptococcus intermedius [6]. Gram-positive and gram-negative isolates from intra-oral odontogenic abscesses have a susceptibility of 96.43% and 80.00% to clindamycin, respectively [7].

2.5. Dosage of clindamycin in treatment of periodontitis:

Studies have shown that the most effective dosage of Clindamycin for periodontal diseases is to give 300 mg three times daily for 10 days [8].

3. STUDIES ON CLINDAMYCIN AS AN ADJUVANT TO PERIODONTAL THERAPY

3.1 Gordon et al

In 1985, Gordon et al assessed the efficacy of clindamycin in the management of refractory patients who had previously been unsuccessfully treated. Microbiologically, Clindamycin affected the subgingival microflora, which had been demonstrated by darkfield microscopy. Clinically, it influenced the progression of periodontitis by improving the periodontal parameters [9]. In 1990, another study by Gordon et al, on thirty adult refractory periodontitis patients, scaling and adjunct clindamycin treatment decreased the incidence of active disease sites per patient [10].

3.2 Walker and Gordon

In 1990, the authors investigated the effect of clindamycin hydrochloride on the microbiota associated with refractory periodontitis, as an adjunct to scaling. Before oral clindamycin therapy, both active and control sites had 50% of spirochetes and motile rods and 40% gram-negative anaerobic rods. Bacteroides intermedius and Porphyromonas gingivalis were higher in the active sites when compared to control sites and accounted for approximately 20% of the cultured microbiota in the former. After administration of oral clindamycin, the gram-negative microbiota was either terminated or overtly decreased. One year after therapy, spirochetes and motile rods were reduced to 15%. Gram-negative anaerobic rods were also reduced to 20%, with Bacteroides intermedius and Porphyromonas gingivalis reduced to 2% of the cultured microbiota from the previously active sites [11].

3.3 Magnusson et al

In 1994, Magnusson et al conducted a study in 21 Refractory periodontitis patients for a period of 2 years. The study population was divided into 3 groups: Group 1 received SRP + Augmentin (Amoxicillin 250, Clavulanic acid 125, thrice daily for 14 days). Group 2 received SRP + clindamycin 150mg four times daily for 10 days, and Group 3 received SRP alone. In groups receiving selective antibiotic therapy + SRP, the percentage of sites showing gain of attachment increased. But during the 2nd year follow-up, the progression of attachment loss in the disease-active sites hadn't completely stopped, with the percentage of sites showing attachment loss decreased from 5.1% to 2.3%, and the proportion of sites showing attachment gain also decreased from 2.0% to 1.0%. The authors suggested that antibiotic therapy plus SRP repeated every 12–15 months may be beneficial for subjects with refractory periodontitis, although it may not completely stop progressive attachment loss [12].

3.4 Eick et al

In an in-vitro study, Eick et al (2000) assessed the effect of clindamycin on the phagocytosing properties of polymorphonuclear leukocytes (PMNs) in GCF obtained from 16 patients with rapidly progressive periodontitis, eight patients with localized juvenile periodontitis, 12 patients with adult periodontitis, and 13 periodontally healthy controls. Porphyromonas gingivalis ATCC 33277 and Aggregatibacter actinomycetemcomitans Tanner FDC 44 were the two test strains used. The addition of clindamycin elevated the percentage of phagocytosing PMNs in all the periodontitis patients, regardless of the test strains used. Enhanced

intracellular killing of both strains was observed in granulocytes of healthy controls if clindamycin was added [13].

3.5 Sigusch et al.

In 2001, they conducted a 2-year study in 48 patients with generalized rapidly progressive periodontitis. The study population was divided into four groups. Group 1 received Scaling and root planing (SRP) + doxycycline (200 mg, once daily, 8days) (N = 12), Group 2 received SRP + Metronidazole (500 mg twice daily for 8 days) (N = 15), Group 3 received SRP + clindamycin (150 mg, four times daily for 8 days) (N = 11) and Group 4 received SRP and no antibiotic was given. Scaling and root planning (SRP) was done in 2 steps for all groups. By 6 months after the second-step SRP, the clindamycin group showed a significantly greater reduction of Probing Pocket depth (PPD) from baseline.

Clindamycin as an adjuvant to SRP induced a greater PPD reduction compared to controls. A significantly high clinical attachment loss (CAL) gain was also noted in the clindamycin group compared with controls. Sulcular Bleeding Index decreased most in the groups receiving metronidazole and clindamycin. Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans were almost completely eradicated in groups receiving metronidazole and clindamycin for two years post SRP [14].

3.6 Gómez-Sandoval JR

Gomez-Sandoval conducted a double-blind, randomized clinical trial (RCT) in 42 chronic periodontitis patients with Type II diabetes mellitus to determine the efficacy of a 7-day regimen of oral clindamycin 300 mg thrice daily compared with oral amoxicillin 500mg + metronidazole 250 mg thrice daily as an adjuvant to SRP. The results showed that one week of administration of clindamycin or amoxicillin/metronidazole exhibited the same efficacy in the reduction of probing depth, plaque index, and bleeding on probing in patients with chronic periodontitis and type II diabetes [15]. Table 1 shows the main features of the studies on clindamycin as an adjuvant to periodontal therapy.

Table 1. Studies on Clindamycin as an adjuvant to periodontal therapy [10-15]

Sno	Author	Population	Results (Post-operative)
1	**Gordon et al (1985)**	Refractory periodontitis patients	Decreased incidence of active disease sites
2	**Walker and Gordon (1990)**	Refractory periodontitis	Reduction in spirochetes, motile rods, Bacteroides intermedius, and Porphyromonas gingivalis
3	**Magnusson et al. 1994**	Refractory periodontitis patients	The percentage of sites showing attachment loss decreased
4	**Eick et al. (2000)**	Rapidly progressive periodontitis, Localized juvenile periodontitis, Adult periodontitis	Elevated percentage of phagocytosing PMNs in all the periodontitis patients
5	**Sigusch et al (2001)**	Generalized rapidly progressive periodontitis.	Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans were almost completely eradicated
6	**Gomez-Sandoval JR (2020)**	Chronic periodontitis patients with Type II diabetes	Reduction of probing depth, plaque index, and bleeding on probing

4. CLINDAMYCIN AS AN IMMUNOMODULATORY DRUG

Clindamycin overtly reduces the release of proinflammatory cytokines like TNF-α and IL-1β, which can cause added destruction to the periodontium when released excessively by triggering microflora. The reduced production of Tumor Necrosis Factor- alpha (TNF-α) and chemokine CXCL-1 is another mechanism mediating the clindamycin inhibitory effect on inflammatory conditions such as periodontitis [16]. Immunoglobulin-G levels of aggressive periodontitis patients were increased when treated with systemic clindamycin as an adjuvant [17].

5. CLINDAMYCIN AS AN LDD AGENT

Normally, clindamycin is a safer drug except for pregnant and lactating mothers [18]. The common side effects of oral clindamycin include vomiting, nausea, diarrhea, and a high risk of Clostridium difficile infection [19]. These side effects can be avoided by administering the drug as LDD. In 2018, clindamycin-loaded complex film, which can be used as LDD in periodontal therapy, was developed. This clindamycin-loaded complex was prepared with chitosan and alginate, with positive in vitro drug content, drug release, and drug adhesion. The clindamycin-loaded polyelectrolyte films that have delayed release, together with high swelling ability and adhesiveness, and high drug content, were formed [20].

In another study, a delayed drug release of more than one week by loading the drug into the chitosan microparticles was achieved. This study showed a positive drug release profile. The results showed that spray-dried clindamycin-loaded microparticles with sustained antimicrobial efficacy appear to be a promising periodontal therapy for drug delivery into the periodontal pocket [21]

DISCUSSION

Clindamycin is a broad-spectrum antimicrobial affecting gram-positive aerobic and anaerobic bacteria, particularly against

bacteria producing beta-lactamase [4]. Clindamycin was the most active antibiotic against Streptococcus constellatus [6]. Clindamycin highly reduces the release of proinflammatory cytokines like TNF-α and interleukin 1-beta (IL-1β) [16]. Enhanced intracellular killing of Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans was observed in granulocytes of healthy controls if clindamycin was added [13]. When given as an adjuvant to SRP, clindamycin is effective in the treatment of refractory periodontitis, a rapidly progressive form of periodontitis, as well as in the management of type II diabetic patients with periodontitis [10-15].

Even though it has many advantages, the only disadvantage associated with systemic use of clindamycin is pseudomembranous colitis due to Clostridium difficile infection [1]. But this can be avoided with proper precautions as well by exploring the possibility of using the drug as LDD.

CONCLUSION

Clindamycin as an adjuvant to periodontal therapy has remarkable success in the management of refractory periodontitis, which showed a healthy microbial shift postoperatively as well as immunomodulatory activity. Thus, Clindamycin can be rightfully called the "Unsung hero of systemic antimicrobials in the management of periodontitis."

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How to cite this article: Muthuraj MSA, Mary Kural Ayeni MAJ. Clindamycin: An Unsung Hero in the management of periodontitis. Indian J Integr Med. 2026; 6(1):31-34.

Funding: None; Conflicts of Interest: None Stated