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Periodontitis: Pathogenesis, Clinical Manifestations, and Advances in Treatment Strategies

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Abstract: Periodontitis is a prevalent chronic inflammatory disease that results in the progressive destruction of the supporting structures of the teeth ultimately leading to tooth loss if untreated. It is caused by the complex interaction between microbial plaque, bacterial biofilms and the host immune-inflammatory response. This review provides a comprehensive overview of the etiology, pathogenesis, clinical features, diagnostic methods, and contemporary treatment strategies of periodontitis. Emphasis is placed on recent advances, including regenerative procedures, host modulation therapies, and novel pharmacologic approaches, highlighting the importance of individualized patient care and evidence-based treatment plans.

Key words: Periodontitis, Pathogenesis, Treatment strategies.

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Introduction

Periodontitis is one of the most widespread multifactorial and progressive oral diseases characterized by the destruction of periodontal ligament, alveolar bone, and connective tissue attachment. According to the World Health Organization it is among the leading causes of tooth loss in adults worldwide in various forms, with severe cases impacting 10–15% of the population (Slots, 2017, Kinane 2017). It not only compromises dental function and aesthetics but also contributes to systemic diseases, including cardiovascular disease, diabetes mellitus, and adverse pregnancy outcomes (Sanz et al., 2018). Understanding the multifactorial nature of periodontitis is key to effective prevention and management (Kwon et al., 2021). The primary etiological factor in periodontitis is the presence of a pathogenic subgingival biofilm composed mainly of Gram-negative anaerobic bacteria such as *Porphyromonas*

gingivalis, *Tannerella forsythia*, and *Treponema denticola* (Hajishengallis, 2015, Bartold 2019). Pathogenesis begins with microbial colonization, leading to gingival inflammation (gingivitis). If untreated, it progresses to periodontitis, marked by epithelial attachment loss, connective tissue destruction, and alveolar bone resorption. Host factors, including genetic predisposition, smoking, systemic diseases (e.g., diabetes), and stress, contribute significantly to disease susceptibility and progression. The immune response, although protective the dysregulated host immune system leads to collateral tissue damage through the release of proinflammatory cytokines (e.g., IL-1β, TNF-α) and matrix metalloproteinases (MMPs), disrupting the balance between tissue destruction and repair. Genetic susceptibility, smoking, metabolic disorders, and stress act as modifying factors.

Clinical manifestations of Periodontitis include: Gingival redness, swelling, and bleeding, deep periodontal pockets (≥4 mm), clinical attachment loss (CAL), tooth mobility and migration, alveolar bone loss observable radiographically, halitosis and discomfort.

The 2018 classification by the American Academy of Periodontology introduced a staging (I-IV) and grading (A-C) system based on severity, complexity, and risk factors (Tonetti et al., 2018).

Comprehensive diagnosis includes: Periodontal charting (probing depth, bleeding on probing, CAL), Radiographs for alveolar bone evaluation, Microbiological assays (if necessary), Biomarker assessment (e.g., MMP-8, IL-1β). Emerging diagnostics involve salivary proteomics and detection tools to improve early diagnosis and monitor therapeutic outcomes.

The cornerstone of periodontitis management is non-surgical therapy.

- **Scaling and root planing (SRP):** The gold standard for initial treatment, aiming to remove subgingival plaque and calculus under local anesthesia. It significantly reduces pocket depth and clinical inflammation (Smiley et al., 2015).
- **Antimicrobial adjuncts:** Topical agents (chlorhexidine, minocycline, doxycycline gels) and systemic antibiotics (amoxicillin + metronidazole) are used for specific cases with aggressive or refractory disease (Wim Teughel 2020).
- When non-surgical approaches fail, surgical techniques such as open flap debridement (OFD) and osseous surgery are used.
- **Open flap debridement (OFD)** provides direct access to deep pockets, while osseous surgery reshapes and contours bone defects.

Regenerative procedures utilize use of bone grafts, guided tissue regeneration (GTR), and enamel matrix derivatives (EMDs) to restore periodontal structures.

Host Modulation Therapy (HMT) targeting the host response has emerged as an adjunct strategy. HMT targets the host response using agents like subantimicrobial dose doxycycline (SDD), which reduces collagen degradation by inhibiting MMPs. NSAIDs and bisphosphonates may also be used, though they are not routinely recommended due to potential side effects (Muhammad Saad Shinwari et al. 2014).

Laser and Photodynamic therapy: Diode, Nd:YAG, and Er:YAG lasers are minimally invasive options used for bacterial reduction and tissue biostimulation. Photodynamic therapy (PDT) utilizes photosensitizers activated by light to eliminate pathogens without contributing to antimicrobial resistance (Ivanaga ET AL., 2019 , Haider A Alwaeli et al., 2015).

Recent studies highlight the role of probiotics, nanotechnology-based drug delivery, stem cell therapy, and personalized medicine in periodontitis management. **Adjunctive and emerging therapies use probiotics that** promote beneficial oral flora balance.. **Nanoparticles** improve drug delivery. **Stem Cell Therapy** shows promise in tissue regeneration but is in experimental stages. **Artificial Intelligence a**ids in diagnosis, prognosis, and personalized treatment planning (Schwendicke et al., 2020).

Long-term success depends on strict supportive periodontal therapy (SPT) every 3–6 months, lifestyle modifications (smoking cessation, glycemic control), and patient compliance. Prognosis varies by stage, grade, and systemic influences.

Table 1. Comparison of Non-Surgical Periodontitis Treatments

Treatment Modality	Key Features	Clinical Outcomes	Reported Efficacy*	Limitations
Scaling and Root Planing (SRP)	Mechanical debridement	↓ PD**, ↓ CAL loss, ↓ bleeding	30–60% PD reduction (11)	Less effective in pockets >6 mm
Topical Antimicrobials	Chlorhexidine, doxycycline gels	↓ localized inflammation	20–40% improvement (13)	Limited duration, resistance risk
Systemic Antibiotics	Amoxicillin + Metronidazole	↓ *P. gingivalis*, ↑ CAL gain	Up to 1 mm additional CAL gain (13)	Risk of resistance, GI side effects
Photodynamic Therapy (PDT)	Photosensitizer + laser light	Antibacterial, ↓ inflammation	30% PD reduction	Costly, used as adjunct only
Probiotics	*L. reuteri*, *B. bifidum* strains	↓ pathogenic bacteria, ↓ inflammation	10–25% improvement	Supportive, not standalone therapy

Table 2. Comparison of Surgical and Regenerative Treatments

Treatment Approach	Application	Clinical Benefits	Reported Efficacy*	Drawbacks
Open Flap Debridement (OFD)	Surgical debridement	Improved access, ↓ PD	2–3 mm PD reduction (12)	Risk of tissue loss
Osseous Surgery	Bone reshaping	Eliminates bony defects, ↓ recurrence	60–70% improvement	Esthetic compromise possible
Guided Tissue Regeneration (GTR)	Barrier membranes for regeneration	↑ Bone fill, ↑ Ligament formation	3–4 mm CAL gain in intrabony defects	Technique-sensitive, exposure risk
Enamel Matrix Derivatives (EMD)	Protein gel for regeneration	↑ Cementum/PDL formation, ↑ Bone fill	60–80% bone regeneration (comparable to GTR)	Expensive, variable response
Stem Cell Therapy (experimental)	MSC-based tissue engineering	Potential for full regeneration	70% bone fill (preclinical trials)	Not yet standard practice

*Reported efficacy based on clinical trial averages for PD (probing depth) and CAL (clinical attachment level) improvements. PD = Probing Depth; CAL = Clinical Attachment Level

Conclusion

Periodontitis is a complex and chronic condition significant public health concern due to its prevalence and systemic implications requiring a multifaceted treatment approach. Early detection, risk assessment, patient education and evidence-based, personalized treatment strategies—including mechanical debridement, host modulation, and regenerative methods are essential to prevent disease progression, tooth loss and crucial for favorable outcomes. Advances in diagnostics, regenerative biology, and

precision medicine promise to reshape future periodontal care. Continuous research is crucial to develop more effective, targeted, and regenerative therapies.

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