Review Article

Bridging Oral Health and Neurodegeneration: The Interplay Between Periodontitis and Parkinson's Disease

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Abstract
Background: Parkinson's disease (PD) and chronic periodontitis are highly prevalent conditions that intersect through systemic inflammatory and neurodegenerative axes. Emerging clinical investigations indicate that severe periodontal disease correlates with an earlier onset of PD and accelerated clinical progression.
Objective: To review the biological pathways linking oral inflammation to central nervous system pathology and evaluate the clinical impact of non-surgical periodontal therapy (NSPT).
Methods: Literature concerning microbial translocation, systemic cytokine priming, protein misfolding, and periodontal interventions was critically reviewed.
Results: Periodontitis is associated with a 2.3-fold greater risk of PD [1]. Pathological mechanisms involve *Porphyromonas gingivalis* translocation and gingipain-mediated alpha-synuclein aggregation [1]. NSPT demonstrates measurable reductions in serum inflammatory markers [2].
Conclusion: Integrating structured periodontal care into multidisciplinary management is essential for mitigating systemic inflammation in PD patients.
Keywords: Parkinson's disease, Periodontitis, *Porphyromonas gingivalis*, Systemic inflammation, Non-surgical periodontal therapy.

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1. INTRODUCTION

Parkinson's disease (PD) is a progressive neurodegenerative disorder affecting over 1 0 million individuals globally, characterized by classic motor deficits such as resting tremor, rigidity, and bradykinesia, alongside diverse non-motor symptoms. Concurrently, chronic periodontitis impacts nearly half of the world's adult population. In recent years, clinicians and researchers have recognized that oral health is intimately connected to systemic wellness. Growing epidemiological data reveal that chronic periodontal disease correlates with an earlier onset of Parkinson's disease—averaging 3.2 years earlier—and faster clinical and cognitive decline, independent of traditional confounders like age or smoking status [1].

2 MECHANISTIC PATHWAYS LINKING ORAL HEALTH TO THE BRAIN

The bridge connecting chronic periodontitis to central nervous system pathology is built upon three intertwined biological mechanisms that amplify neurodegeneration:

- **Microbial Translocation:** Keystone periodontal pathogens, most notably *Porphyromonas gingivalis*, possess remarkable neuroinvasive properties. Studies identifying bacterial components in postmortem brain tissues highlight their ability to travel from inflamed periodontal pockets into systemic circulation and across the blood-brain barrier [1].

- **Systemic Inflammatory Priming:** Chronic periodontal inflammation constantly triggers peripheral immune cells, releasing pro-inflammatory cytokines into the bloodstream. These mediators elevate baseline systemic inflammation, predisposing cerebral microglia to a heightened state of activation. Documented plasma comparisons between PD patients with and without periodontitis show pronounced disparities:

INTERLEUKIN-6 (IL-6)	TUMOR NECROSIS FACTOR-ALPHA
41.2 pg/mL (vs 28.7 pg/mL) [1]	**35.6 pg/mL** (vs 22.1 pg/mL) [1]

Protein Misfolding and Aggregation: Bacterial proteases known as gingipains directly promote alpha-synuclein aggregation. By cleaving proteins at specific amino acid residues, these enzymes accelerate fibril formation over twofold, induce severe cellular oxidative stress, and impair neuronal lysosomal function [1].

3. CLINICAL EVIDENCE AND INTERVENTIONAL INSIGHTS

Large-scale epidemiological studies reinforce these biological mechanisms. Long-term cohort analyses (*n* = 12,140) have demonstrated that individuals with severe periodontitis face a significantly elevated risk of developing neurodegenerative conditions, while accumulated tooth loss correlates with a 1.7-fold increased incidence of PD [1].

Intervention Approach	Documented Clinical Outcome	Duration / Persistence
Non-Surgical Periodontal Therapy (NSPT)	34% reduction in circulating serum IL-6 levels [2]	Sustained across 3–6 months [2]
Ultrasonic Debridement & Scaling	2.1-point improvement in clinical motor scores [2]	Sustained across 3–6 months [2]
Adjunctive Antimicrobial Protocols	Effective reduction of subgingival bacterial pathogen load [2]	Short- to medium-term [2]

4 THERAPEUTIC IMPLICATIONS IN MULTIDISCIPLINARY CARE

Recognizing periodontitis as an active contributor to systemic inflammation shifts its management from a purely dental concern to an essential component of comprehensive neurodegenerative care. Clinical guidelines now emphasize several proactive strategies:

- **Comprehensive Periodontal Evaluations:** Establishing routine, annual periodontal charting and assessments for all patients diagnosed with Parkinson's disease [2].
- **Proactive Periodontal Maintenance:** Implementing regular ultrasonic debridement and subgingival scaling every 3 to 6 months to suppress systemic inflammatory flare-ups [2].
- **Targeted Antimicrobial Interventions:** Utilizing localized or adjunctive antimicrobial therapies for patients presenting with deep periodontal pockets (> 5 mm) [2].

5 FUTURE RESEARCH DIRECTIONS

Moving forward, clinical research must focus on well-powered randomized controlled trials evaluating motor and cognitive endpoints following structured periodontal treatments. Promising avenues include phase III trials of selective gingipain inhibitors and the integration of salivary biomarker panels for early risk stratification [2].

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