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THE ROLE OF OTORHINOLARYNGOLOGIC DISEASES IN THE ETIOPATHOGENESIS OF INFLAMMATORY PERIODONTAL PATHOLOGY IN ADULTS: INTERDISCIPLINARY ASPECTS

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Abstract

This paper provides a detailed analysis of the combined pathogenic mechanisms linking chronic, focal otorhinolaryngologic pathologies (such as chronic rhinosinusitis, tonsillitis, and various etiologies of impaired nasal breathing) to the initiation and progression of chronic generalized periodontitis in adult patients. Drawing on contemporary clinical data, we examine the aerodynamic, microbiological, and immunologic triggers that drive this mutual aggravation. The findings demonstrate that persistent upper respiratory tract infections in adults induce a stubborn local dysbiosis within the oral cavity and a systemic cytokine imbalance that accelerates alveolar bone resorption. Ultimately, this paper outlines and supports a mandatory interdisciplinary framework for managing these patients through coordinated dental and ENT interventions.

Keywords: inflammatory periodontal diseases, chronic periodontitis, otorhinolaryngology, rhinosinusitis, mouth breathing, oral dysbiosis, systemic inflammatory response.

Introduction

Inflammatory and destructive periodontal diseases in adults remain among the most complex, multifactorial, and socially significant challenges in modern dentistry. According to the World Health Organization (WHO), various signs of periodontal pathology—ranging from bleeding gums to severe destruction of the supporting apparatus and subsequent tooth loss—are observed in more than 90% of the adult population worldwide over the age of 35. Despite continuous advancements in local therapeutic and surgical modalities, clinicians routinely face high recurrence rates and cases of chronic periodontitis that are stubborn and resistant to standard care protocols.

The current paradigm in periodontology views the periodontal tissues as a highly sensitive ecosystem that reacts sharply to homeostatic shifts in adjacent anatomical regions and the body as a whole. Among the comorbid conditions capable of acting as triggers or modifiers of this inflammatory process, chronic pathology of the ENT organs holds a prominent place. Chronic rhinosinusitis, deviated nasal septa, polyposis, and decompensated tonsillitis are remarkably prevalent among working-age individuals.

The close anatomical proximity, shared lymphatic drainage, microcirculatory networks, and innervation naturally forge strong pathogenic links between the ENT tract and the periodontal

complex. In adult patients, these connections are further aggravated by a long, often decades-long history of ENT disorders, which eventually exhausts the host's compensatory reserves. To date, however, comprehensive diagnostic and treatment algorithms for patients with concurrent periodontal and upper respiratory pathologies remain poorly systematized. This gap in care highlights the relevance of the current study.

Pathogenic Mechanisms of Comorbid Association

The interplay between inflammation in the nasopharynx and the periodontal tissues in adults operates through three primary pathogenic pathways:

1. Mouth Breathing and Local Xerostomic Shifts

Persistent impairment of nasal breathing in adults—whether caused by a deviated septum, hypertrophic rhinitis, or sinonasal polyposis—forces a transition to oral or mixed breathing. In the adult organism, this shift triggers several localized pathological changes:

- **Chronic mucosal dehydration:** The constant intake of unconditioned air through the oral cavity dries out the marginal gingiva and alveolar mucosa. This process thins the protective mucin layer, causes epithelial desquamation, and exposes receptors, leaving the tissues vulnerable to mechanical and microbial irritants.
- **Qualitative and quantitative salivary deficits:** Mouth breathing triggers hyposalivation, dropping salivary flow rates below 0.3 mL/min. As a result, saliva loses its protective properties, showing marked decreases in lysozyme, lactoferrin, and secretory immunoglobulin A (sIgA). The buffering capacity of the oral fluid plummets, causing a steady shift toward an acidic pH. This acidic environment catalyzes the mineralization of subgingival dental plaque and accelerates the maturation of aggressive biofilms.
- **Microcirculatory disturbances:** Altered breathing dynamics and a constantly half-open mouth distort the hydrodynamic equilibrium in the periodontal capillary bed. The resulting chronic tissue hypoxia leads to venular dilation, blood stasis, and lipid peroxidation (LPO) within the gingival tissues.

2. Microbial Translocation and Pathogen Synergism

The oral cavity, tonsils, and paranasal sinuses form a shared biotope in adults, characterized by a constant exchange of microflora. Chronic infectious foci in the ENT organs (especially purulent sinusitis and decompensated tonsillitis) create a permanent reservoir for highly virulent strains like Staphylococcus\,aureus, Streptococcus\,pneumoniae, and Pseudomonas\,aeruginosa.

- **Post-nasal drip syndrome:** Infected exudate from the nasopharynx regularly drains into the oral cavity, mostly at night, settling in the retromolar areas and marginal gingiva.
- **Creation of anaerobic niches:** Tissue hypoxia driven by mouth breathing creates ideal conditions for the selection and proliferation of obligate periodontopathogens of the "red complex" (Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola). A microbial synergism emerges between ENT pathogens and periodontopathogens, sharply increasing the expression of virulence factors and speeding up the destruction of the periodontal ligament.

3. Systemic Immunopathological Alteration and the Cytokine Cascade

A long-standing purulent-inflammatory focus in the upper respiratory tract sensitizes the body and alters the immune response:

- **Endogenous intoxication:** Bacterial lipopolysaccharides (endotoxins) continuously spill from ENT foci into the systemic circulation. They activate macrophages and

monocytes, triggering an overproduction of pro-inflammatory mediators, including interleukins (IL\text{-}1\beta, IL\text{-}6), tumor necrosis factor (TNF\text{-}\alpha), and matrix metalloproteinases (MMP\text{-}1, MMP\text{-}8).

- **Osteoclastic resorption:** This excess of pro-inflammatory cytokines reaches the periodontal tissues via the microcirculatory network, where it activates the RANKL system (receptor activator of nuclear factor kappa-B ligand). This activation drives osteoclastogenesis, causing rapid destruction of the interdental septa and accelerating alveolar bone loss.

Materials and Methods

The clinical and laboratory data for this study were gathered from a comprehensive evaluation of 140 patients aged 30 to 55 years, all diagnosed with moderate-to-severe chronic generalized periodontitis.

The cohort was divided into three distinct clinical groups:

1. **Group I (Main Group, n = 55):** Patients presenting with chronic generalized periodontitis coupled with confirmed, chronic ENT pathologies (chronic purulent or polypous rhinosinusitis, deviated nasal septum with persistent airway obstruction, or subcompensated/decompensated chronic tonsillitis).
2. **Group II (Comparison Group, n = 55):** Patients with a similar severity of periodontitis but with healthy, unobstructed nasal breathing and no clinical history of ENT disorders.
3. **Group III (Control Group, n = 30):** Periodontally and systemically healthy volunteers with normal nasal breathing function.

The diagnostic framework included:

- **Periodontal assessment:** Evaluation of oral hygiene via the OHI-S index (Green-Vermillion), Russell's Periodontal Index (PI), and the Papillary-Marginal-Gingival Index (PMA) as modified by Parma. Periodontal pocket depth (PPD) and clinical attachment loss (CAL) were measured using a graduated periodontal probe. Bone destruction was quantified via Cone-Beam Computed Tomography (CBCT).
- **ENT assessment:** Anterior and posterior rhinoscopy, nasal and sinus endoscopy, evaluation of nasal airflow (anterior active rhinomanometry), and pharyngoscopy.
- **Laboratory and Statistical testing:** Assessment of salivary flow rates and pH levels of the oral fluid. Statistical analysis was performed using SPSS Statistics 26.0. Student's t-test and the non-parametric Mann-Whitney U test were applied to determine statistical significance, with thresholds set at $p < 0.05$.

Results and Discussion

The findings revealed a direct correlation between the severity of ENT pathologies and the intensity of destructive periodontal processes in adults.

Clinical Marker	Group I (With ENT)	Group II (No ENT)
Mean Pocket Depth (PPD, mm)	5.2 ± 0.4*	3.8 ± 0.3
PMA Index (%)	58.4 ± 4.2%*	36.1 ± 3.5%
Salivary Flow Rate (mL/min)	0.24 ± 0.03*	0.48 ± 0.05
Salivary pH	6.22 ± 0.12*	6.95 ± 0.15

* $p < 0.05$ compared to Group II.

Patients in Group I exhibited a notably aggressive form of periodontitis. The mean periodontal pocket depth in individuals with concurrent ENT pathologies reached 5.2 ± 0.4 mm, which was significantly deeper than that observed in the comparison group (3.8 ± 0.3 mm, $p < 0.05$). The PMA index in the main group reached 58.4 ± 4.2%, pointing to intense exudative inflammation of the marginal gingiva.

The patterns of bone loss seen on CBCT scans were especially telling. Patients with a long history of mouth breathing showed a vertical pattern of alveolar bone resorption in the anterior segments, leading to deep infrbony pockets. This finding aligns perfectly with the theory of localized mechanical and ischemic tissue injury caused by the drying effect of continuous airflow.

Evaluation of the oral fluid in individuals with chronic sinusitis showed a critical drop in salivary flow rates down to 0.24 ± 0.03 mL/min (compared to 0.48 ± 0.05 mL/min in the comparison group), with the pH shifting significantly toward acidosis (6.22 ± 0.12). Furthermore, microbial cultures from deep periodontal pockets in patients with decompensated tonsillitis yielded Staphylococcus aureus and hemolytic streptococci in 64.7% of cases—matching the pathogens verified in their tonsillar lacunae. This provides undeniable proof of bacterial translocation and continuous re-infection.

Conclusion

1. Chronic otorhinolaryngologic diseases in adult patients serve as a potent comorbid factor. They exacerbate destructive processes within the periodontium, shifting the inflammation into an aggressive state that resists conventional therapies.

2. The core links connecting ENT pathologies to chronic periodontitis in adults include local hyposalivation, oral fluid acidosis driven by mouth breathing, bacterial translocation from upper respiratory infectious foci, and a systemic cytokine imbalance that fuels osteoclastic bone resorption.

3. Successful rehabilitation of adult patients suffering from chronic generalized periodontitis alongside upper respiratory tract issues is impossible without an interdisciplinary protocol. Periodontal interventions (including scaling, root planing, and flap surgeries) must be performed in tandem with surgical or conservative ENT clearance to restore proper nasal breathing.

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