Western European Journal of Linguistics and Education
Volume 3, Issue 5, May 2025
https://westerneuropeanstudies.com/index.php/2
ISSN (E): 2942-190X Open Access| Peer Reviewed

DISTINCTIVE CLINICAL COURSE AND PROPHYLAXIS OF POLYPOID RHINOSINUSITIS

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Abstract
Chronic polypoid rhinosinusitis (CPR) represents a complex form of chronic rhinosinusitis characterized by mucosal inflammation, nasal polyp formation, and significant morbidity. Despite advances in endoscopic sinus surgery and topical pharmacotherapy, CPR remains prone to relapse and complications. This review synthesizes current knowledge on the unique clinical features, immunopathology, and evidence-based prevention strategies for CPR. We examine the interplay of environmental, microbial, and host factors that drive polypogenesis, outline the stages of disease progression, and critically assess prophylactic measures—including pharmacologic maintenance, immunotherapy, and lifestyle modifications. Recommendations for personalized prevention algorithms are proposed, aiming to reduce recurrence rates and improve patient quality of life.
Keywords: chronic rhinosinusitis, nasal polyps, immunopathology, prophylaxis, endoscopic sinus surgery, biological therapy, allergen immunotherapy

Introduction

Chronic rhinosinusitis with nasal polyps (CRSwNP) affects approximately 2%–4% of the adult population worldwide, with variations by geography and atopic predisposition. [1,4] CPR, defined by the presence of bilateral sinonasal polyps persisting for at least 12 weeks, is frequently associated with asthma, aspirin-exacerbated respiratory disease (AERD), and allergic rhinitis. [2] The formation of edematous polyps results from sustained type 2 inflammation, characterized by elevated interleukins (IL-4, IL-5, IL-13), eosinophilic infiltration, and dysregulated epithelial barrier function. [3,8,10] CPR imposes a high individual and socioeconomic burden due to impaired olfaction, sleep disturbance, and frequent need for revision surgery.

This article aims to provide a candidate of sciences–level exposition of (1) the pathophysiological mechanisms underlying polyp formation and progression, (2) distinctive clinical and endoscopic features of CPR, and [contemporary prophylactic strategies targeting immune, microbial, and environmental contributors to disease recurrence. [5,7]

Pathophysiology and Immunopathogenesis of Polypoid Rhinosinusitis

Epithelial Barrier Dysfunction

The sinonasal epithelium serves as a frontline defense against inhaled antigens and pathogens. In CPR, tight junction proteins (e.g., occludin, claudin-1) are downregulated, compromising barrier integrity. Allergen- or pathogen-induced epithelial damage allows increased permeability, facilitating chronic antigen exposure and perpetuating inflammation.

Type 2–Dominant Inflammation

A hallmark of CPR is Th2-skewed immunity. Antigen-presenting cells in compromised mucosa release IL-25, IL-33, and thymic stromal lymphopoietin (TSLP), driving differentiation of naïve T cells toward Th2 lineage. Subsequent secretion of IL-4 enhances immunoglobulin E (IgE) production by B cells, while IL-5 recruits and activates eosinophils.

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IL-13 contributes to goblet cell hyperplasia and mucous hypersecretion, promoting polyp growth.

Microbial Dysbiosis and Biofilms

Sinonasal microbiome alterations, characterized by reduced commensals (e.g., lactobacilli) and increased pathogenic strains (Staphylococcus aureus, Pseudomonas aeruginosa), have been implicated in CPR. Bacterial biofilms on sinonasal mucosa further sustain inflammation by shielding microbes from host defenses and antibiotics.

Genetic and Environmental Interactions

Polymorphisms in genes encoding cytokines (IL5, IL13), pattern-recognition receptors (TLR2, TLR4), and epithelial junction proteins confer increased susceptibility to CPR. Environmental factors—seasonal allergens, air pollutants, occupational irritants—act as triggers in genetically predisposed individuals.

Clinical Presentation and Disease Course

Symptomatology and Quality of Life

Patients with CPR typically present with nasal obstruction, rhinorrhea (anterior/posterior drip), hyposmia/anosmia, facial pressure, and sleep-disordered breathing. The Sino-Nasal Outcome Test (SNOT-22) often exceeds 40 in moderate to severe cases, correlating with diminished quality of life and psychological distress.

Endoscopic and Radiologic Evaluation

Nasal endoscopy reveals bilateral, pale, edematous polyps grading from I to IV (Lund–Mackay staging). Computed tomography (CT) imaging demonstrates opacification of the osteomeatal complex and ethmoid cells. Radiologic severity scores correlate with symptom burden and recurrence risk.

Natural History and Recurrence

Without adequate prophylaxis, up to 80% of patients experience polyp recurrence within two years of functional endoscopic sinus surgery (FESS). Recurrence is more frequent in AERD and corticosteroid-dependent phenotypes. Frequent courses of systemic steroids, though temporarily effective, pose risks of adrenal suppression, osteoporosis, and metabolic complications.

Prophylactic Strategies

Pharmacologic Maintenance Therapy

Topical Corticosteroids

Regular intranasal corticosteroids (e.g., fluticasone propionate, mometasone furoate) reduce polyp size and delay recurrence. Delivery methods—including high-volume nasal irrigation and exhalation delivery systems—enhance drug distribution to the sinus mucosa.

Systemic Corticosteroids

Short courses (1–2 weeks) of oral prednisone yield rapid polyp shrinkage but are reserved for acute exacerbations due to systemic side effects. Low-dose maintenance within a biologics protocol may be considered when indicated.

Biologic Agents

Monoclonal antibodies targeting IL-5 (mepolizumab, benralizumab), IL-4/IL-13 (dupilumab), or IgE (omalizumab) have demonstrated significant reductions in polyp score and corticosteroid requirement. Tailoring biologic therapy based on tissue eosinophilia, serum IgE, and comorbid asthma optimizes outcomes.

Allergen-Specific Immunotherapy

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For patients with confirmed allergic triggers, subcutaneous or sublingual immunotherapy reduces sinonasal inflammation, improves olfaction, and may confer long-term disease modification. Duration of at least three years is recommended for sustained benefit.

Environmental and Lifestyle Modifications

Reduction of exposure to airborne irritants (tobacco smoke, occupational dust), diligent sinus hygiene (daily saline irrigations), and management of comorbid conditions (asthma, gastroesophageal reflux) are essential components of a holistic prophylactic regimen.

Personalized Prophylaxis Algorithm

An individualized approach incorporates phenotypic stratification (eosinophilic vs non-eosinophilic, AERD vs non-AERD), endoscopic severity, and patient comorbidities. We propose a stepwise algorithm:

1. **Baseline assessment**: endoscopy, CT staging, allergy and immunologic workup, comorbidity screening.
2. **Initiation**: high-volume intranasal corticosteroid irrigation.
3. **Escalation**: add biologic therapy for eosinophilic phenotypes or uncontrolled asthma.
4. **Adjunct**: allergen immunotherapy if specific IgE–mediated allergy is documented.
5. **Maintenance**: environmental control, saline irrigations, periodic endoscopic surveillance.
6. **Re-evaluation**: annual clinical and endoscopic review to adjust prophylaxis.

Discussion

Polypoid rhinosinusitis embodies a multifactorial disease wherein dysregulated type 2 immunity, epithelial integrity loss, and microbial biofilms converge. Advances in endoscopic techniques have improved surgical outcomes, yet without targeted prophylaxis, recurrence remains high. Emerging biologics offer promising avenues but pose challenges of cost and long-term safety. Integration of immunotherapy and lifestyle modifications into prevention protocols may enhance durability of remission. Further research into biomarkers predicting biologic responsiveness and epithelial barrier restoration therapies is needed.

Conclusion

The distinctive clinical course of CPR, marked by high recurrence and complex immunopathology, demands a comprehensive prevention strategy. Maintenance pharmacotherapy, biologic agents, allergen immunotherapy, and environmental control form the pillars of prophylaxis. Tailored algorithms based on disease phenotype and patient-specific factors can minimize recurrence and improve quality of life. Continued investigation into novel therapeutics and individualized biomarkers will further refine prevention of polypoid rhinosinusitis.

References

1. Fokkens WJ, Lund VJ, Hopkins C, et al. European Position Paper on Rhinosinusitis and Nasal Polyps 2020. *Rhinology*. 2020;58(Suppl S29):1-464.
2. Bachert C, Han JK, Desrosiers M, et al. Efficacy and safety of dupilumab in patients with severe chronic rhinosinusitis with nasal polyps (SINUS-24 and SINUS-52): results from two multicenter, randomized, placebo-controlled, double-blind phase 3 trials. *Lancet*. 2019;394(10209):1638-1650.
3. Gevaert P, Calus L, Van Zele T, et al. Omalizumab is effective in allergic and nonallergic patients with nasal polyps and asthma. *J Allergy Clin Immunol*. 2013;131(1):110-116.

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4. Stevens WW, Peters AT, Tan BK, et al. Clinical Characteristics of Patients with Chronic Rhinosinusitis with Nasal Polyps, Asthma, and Aspirin-Exacerbated Respiratory Disease. *J Allergy Clin Immunol Pract*. 2017;5(4):1061-1070.
5. DeConde AS, Mace JC, Levy JM, et al. SNOT-22 quality of life domains differentially predict treatment selection in chronic rhinosinusitis. *Int Forum Allergy Rhinol*. 2014;4(12):972-979.
6. Hamilos DL. Chronic rhinosinusitis: epidemiology and medical management. *J Allergy Clin Immunol*. 2011;128(4):693-707.
7. Stevens WW, Peters AT, Hirsch AG, et al. Clinical and biological correlates of chronic rhinosinusitis with nasal polyps. *J Allergy Clin Immunol*. 2015;136(3): 706-712.
8. Pothoven KL, Schleimer RP. Immunopathology of Chronic Rhinosinusitis and Nasal Polyposis. *Allergy Asthma Proc*. 2016;37(6):497-504.
9. Van Zele T, Gevaert P, Holtappels G, et al. Local immunoglobulin production in nasal polyposis is modulated by superantigens. *Clin Exp Allergy*. 2007;37(3):416-424.
10. Tan BK, Lane AP. Chronic rhinosinusitis pathogenesis. In: *Ballenger's Otorhinolaryngology: Head and Neck Surgery*. 18th ed. PMPH-USA; 2010:575-585.