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PEDIATRIC RHINOSINUSITIS: BIOCHEMICAL AND IMMUNOPATHOGENETIC BASES OF DIAGNOSIS AND TREATMENT

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Abstract

Pediatric rhinosinusitis is a prevalent upper respiratory disorder marked by inflammation of the nasal mucosa and paranasal sinuses. Its pathogenesis involves viral and bacterial infections, allergic reactions, anatomical factors, and biochemical inflammatory mechanisms. Key mediators, including pro-inflammatory cytokines (IL-1β, IL-6, TNF-α), chemokines (IL-8, MCP-1), reactive oxygen species (ROS), and microbial biofilms, sustain inflammation and contribute to chronicity and complications. Current diagnostic strategies combine clinical assessment, imaging, and biochemical marker analysis, facilitating evaluation of disease severity and risk of chronic progression. Effective management requires a multidisciplinary approach, integrating anti-inflammatory and antibacterial therapy, antioxidant support, allergy control, and surgical intervention when needed. Incorporating clinical and biochemical insights enables personalized treatment, optimizing outcomes and reducing relapses and complications.

Keywords

Pediatric rhinosinusitis, biochemical markers, cytokines, chemokines, reactive oxygen species, microbial biofilms, diagnosis, therapy, chronic inflammation

Pediatric rhinosinusitis is one of the most commonly encountered disorders of the upper respiratory tract and has a significant impact on a child's overall health, well-being, and quality of life [2,6,7]. According to various epidemiological studies, up to 5–10% of children under the age of 12 experience acute or chronic inflammation of the paranasal sinuses each year, leading to frequent consultations with pediatricians and otorhinolaryngologists [2,6]. Despite its high prevalence, timely and accurate diagnosis, selection of effective therapeutic strategies, and prevention of potential complications remain pressing and significant challenges in contemporary pediatric otorhinolaryngology [4,7].

Current understanding of the pathogenesis of pediatric rhinosinusitis emphasizes the multifactorial nature of the disease, which involves infectious, allergic, anatomical, and immunological components [3,7,8]. Viral and bacterial pathogens play a central role in initiating inflammation, causing damage to the mucosal epithelium, impairing mucociliary clearance, and triggering the activation of local immune responses [6,7]. At the same time, growing attention is being directed toward the biochemical mechanisms underlying inflammation, including the production of pro-inflammatory cytokines (IL-1β, IL-6, TNF-α), chemokines (IL-8, MCP-1), and activation of NF-κB signaling pathways, all of which contribute to prolonged and chronic inflammation of the nasal and paranasal sinus mucosa [7,9].

Microbial biofilms have gained particular importance in the contemporary understanding of pediatric rhinosinusitis, as they not only contribute to the persistence and resistance of infections to antibacterial therapy but also sustain continuous stimulation of inflammatory responses at the molecular level [5,7,9]. The interplay between infectious agents and allergic or immunological

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components creates a synergistic effect, promoting the chronicity of rhinosinusitis and the development of complicated forms of the disease [3,7].

Investigation of the biochemical and pathogenetic aspects of pediatric rhinosinusitis provides new opportunities to improve diagnostic and therapeutic approaches [7,9]. Identification of molecular markers of inflammation, as well as the study of cytokines, allergic mediators, and biofilms, enables clinicians not only to assess the severity of the disease more accurately but also to develop individualized treatment strategies aimed at preventing chronic progression [7].

Thus, a modern approach to managing rhinosinusitis in children requires the integration of clinical findings with biochemical and pathogenetic mechanisms, allowing for more precise prediction of disease course and optimization of therapeutic interventions [7,8].

Rhinosinusitis in children represents a multifactorial disorder of the upper respiratory tract, characterized by inflammation of the nasal and paranasal sinus mucosa [2,6,7]. The etiology of the condition includes viral infections (such as rhinoviruses, adenoviruses, and influenza viruses), bacterial superinfections (including *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*), allergic reactions, and anatomical features of the pediatric airway [2,6,7].

Biochemical mechanisms of inflammation play a central role in the pathogenesis of rhinosinusitis. Viral infections cause epithelial damage and impair mucociliary clearance, which is accompanied by activation of pro-inflammatory cytokines, including IL-1β, IL-6, and TNF-α, as well as chemokines such as IL-8 and MCP-1 [7,9]. These signaling molecules recruit neutrophils and monocytes to the site of inflammation, amplifying local inflammatory responses and facilitating bacterial superinfection [7]. Bacterial pathogens further release toxic proteins and enzymes that damage the mucosa and promote chronic inflammation [5,7].

The study of immunopathogenetic and biochemical markers in rhinosinusitis allows for a deeper understanding of the molecular mechanisms of inflammation and helps assess disease severity in children [7,9]. Key markers include pro-inflammatory cytokines, chemokines, allergy mediators, and indicators of oxidative stress, all of which regulate both local and systemic immune responses [7].

Pro-inflammatory cytokines, such as IL-1β, IL-6, and TNF-α, play crucial roles in initiating and sustaining the inflammatory process [7]. IL-1β activates leukocytes and induces the production of additional cytokines, thereby amplifying the inflammatory cascade [7]. IL-6 participates in systemic inflammatory responses and stimulates the synthesis of C-reactive protein (CRP), which can be used as a biomarker to monitor disease activity [7,9]. TNF-α increases vascular permeability and contributes to mucosal edema, creating conditions favorable for stagnation of infection [7].

Chemokines, including IL-8 and MCP-1, direct the migration of neutrophils and monocytes to the inflammatory site [7,9]. This targeted recruitment of leukocytes enhances local inflammation, contributes to epithelial barrier disruption, and supports the formation of chronic foci of infection [7].

Particular attention is given to oxidative stress, characterized by excessive production of reactive oxygen species (ROS) [7]. ROS damage epithelial cell membranes and cilia, disrupt mucociliary clearance, and stimulate NF-κB activation, which further promotes the production of pro-inflammatory cytokines [7]. An imbalance between pro-oxidant and antioxidant systems contributes to the prolonged course of inflammation and reduces the regenerative potential of the mucosa [7].

Microbial biofilms represent an additional source of inflammatory stimulation [5,7,9]. They protect bacteria from antibiotics and host immune responses while actively secreting signaling

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molecules that induce cytokine and chemokine production, sustaining chronic inflammation and promoting disease recurrence [5,9].

A comprehensive analysis of these markers allows clinicians to assess the severity of the inflammatory process, predict the risk of rhinosinusitis chronicity, and adjust therapeutic strategies accordingly [7,9]. Modern treatment approaches encompass not only antimicrobial and anti-inflammatory agents but also targeted interventions directed at specific biochemical and immune pathways, opening new prospects for personalized medicine in pediatric otorhinolaryngology [7].

The clinical presentation of rhinosinusitis in children is diverse and depends on the child's age, the etiological factor, and the severity of inflammation. Common symptoms include nasal congestion, mucopurulent discharge, cough, reduced sense of smell, nighttime sleep disturbances, and general malaise [6,8]. In chronic cases, additional manifestations such as headaches, fatigue, decreased appetite, and recurrent episodes of fever may occur [3,4].

From a biochemical perspective, the severity of symptoms often correlates with elevated levels of pro-inflammatory cytokines (IL-1β, IL-6, TNF-α) and chemokines (IL-8, MCP-1). Increased concentrations of these mediators are associated with mucosal edema, excessive mucus production, and enhanced vascular permeability, all of which directly influence the intensity of clinical manifestations. For example, IL-6 functions not only as a systemic marker of inflammation but also correlates with the severity of fever and overall malaise. TNF-α and reactive oxygen species (ROS) contribute to tissue swelling and pain, while IL-8 reflects neutrophil-driven inflammation and susceptibility to bacterial superinfection.

Diagnosis of pediatric rhinosinusitis requires a comprehensive, multifaceted approach. Clinical assessment includes history taking, physical examination, and nasal endoscopy to detect edema, hyperemia, and purulent secretions. Imaging techniques—such as ultrasonography and computed tomography (CT) of the paranasal sinuses—provide precise information on anatomical features and the extent of inflammation [1,4].

Modern diagnostic approaches emphasize the importance of biochemical markers, which can help assess the activity of the inflammatory process and predict disease course. Levels of IL-6, TNF-α, and CRP may serve as indicators of acute disease severity and the effectiveness of antibacterial or anti-inflammatory therapy. Additionally, evaluation of oxidative stress and ROS activity helps identify patients at higher risk of chronic disease progression.

Particular attention is given to detecting microbial biofilms using contemporary laboratory techniques. Biofilms contribute to the persistence of infection despite standard therapy and sustain chronic inflammation. Identifying biofilms can influence treatment strategies, including the need for combined antibacterial therapy, biofilm-disrupting methods, or topical antimicrobial agents. Modern diagnostic protocols also include the measurement of inflammatory biomarkers (IL-6, TNF-α, CRP), allowing assessment of disease severity and therapeutic efficacy [9].

Thus, integrating clinical symptoms, imaging results, and biochemical markers not only clarifies the diagnosis but also enables assessment of disease severity, prediction of progression, and individualization of treatment strategies. This integrated approach allows early identification of children at risk for chronic rhinosinusitis, ensuring timely intervention, minimizing complications, and improving patient quality of life.

Effective treatment of pediatric rhinosinusitis requires a comprehensive approach that addresses clinical manifestations, underlying etiology, and the biochemical and pathogenetic mechanisms of inflammation. The primary therapeutic goals are to reduce the inflammatory response, restore mucociliary clearance, eliminate bacterial infection, and prevent disease chronicity [6,7].

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The management of pediatric rhinosinusitis requires a comprehensive approach that addresses inflammation, infection, oxidative stress, and any underlying allergic or anatomical factors. Anti-inflammatory therapy constitutes a cornerstone of treatment, with topical and systemic corticosteroids effectively reducing the expression of proinflammatory cytokines, including IL-1β, IL-6, and TNF-α, alleviating mucosal edema, and improving sinus drainage. This modulation of the inflammatory cascade has been shown to decrease the levels of chemokines such as IL-8 and MCP-1, thereby limiting neutrophil recruitment and mitigating local tissue damage. Concurrently, when bacterial superinfection is present, targeted antibiotic therapy is employed, tailored to the local microbial flora and pathogen sensitivity. The presence of microbial biofilms, which protect bacteria from immune responses and antibiotics, may necessitate prolonged or combination regimens to disrupt these structures and enhance antimicrobial efficacy.

Supportive interventions complement pharmacological therapy by restoring mucociliary clearance and reducing inflammatory mediators. Nasal saline irrigation facilitates the removal of mucus, debris, and reactive oxygen species (ROS), thereby enhancing the effectiveness of anti-inflammatory and antimicrobial treatments. Short-term use of nasal decongestants can provide symptomatic relief by reducing mucosal swelling, although they do not directly alter the biochemical pathways driving inflammation. Given the role of oxidative stress in disease pathogenesis, antioxidant supplementation—either pharmacological or dietary—may further protect epithelial cells, normalize the redox balance of the mucosa, and attenuate ROS-mediated cytokine activation.

In children with concomitant allergic disorders, antihistamines and desensitization strategies help reduce the release of histamines, leukotrienes, and prostaglandins, decreasing mucosal edema and excessive mucus production, and thereby supporting overall therapeutic efficacy. For chronic or recurrent cases, particularly those associated with anatomical anomalies or persistent biofilms, surgical intervention—including functional endoscopic sinus surgery or adenoidectomy—may be indicated. These procedures aim to restore sinus ventilation and drainage, lower local inflammatory burden, and reduce cytokine-driven biochemical stimulation.

By integrating anti-inflammatory, antimicrobial, supportive, antioxidant, allergological, and surgical strategies, clinicians can achieve comprehensive control of pediatric rhinosinusitis. This approach not only mitigates acute symptoms but also addresses the underlying pathophysiological mechanisms, reduces the risk of chronicity, and enhances long-term clinical outcomes.

Personalized therapy based on the assessment of inflammatory biochemical markers allows for the selection of the most appropriate combination of medications and treatment modalities, minimizing the risk of chronicity and recurrence. A comprehensive therapeutic approach that integrates anti-inflammatory, antimicrobial, antioxidant, and allergy-modulating interventions targets the key pathogenetic mechanisms of pediatric rhinosinusitis, enhancing treatment efficacy and improving patients' quality of life.

Thus, modern strategies for managing rhinosinusitis in children combine clinical evaluation with biochemical and immunopathogenetic analyses, enabling the implementation of individualized treatment plans and reducing the likelihood of progression to chronic disease.

Conclusion. Pediatric rhinosinusitis represents a complex, multifactorial pathology of the upper respiratory tract, in which infectious, immunological, allergic, and biochemical mechanisms play a pivotal role. Viral and bacterial infections initiate epithelial damage, disrupt mucociliary clearance, and activate a cascade of inflammatory responses. Proinflammatory cytokines (IL-1β, IL-6, TNF-α), chemokines (IL-8, MCP-1), reactive oxygen species (ROS), and microbial biofilms collectively create a persistent inflammatory microenvironment, promoting chronicity

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and predisposing to bacterial superinfection. The synergistic interaction between these pathogenic factors further exacerbates the severity of the disease and complicates therapeutic management.

Contemporary diagnostic strategies for pediatric rhinosinusitis integrate clinical evaluation, instrumental imaging, and assessment of biochemical inflammatory markers, allowing for precise determination of disease severity, prediction of its progression, and identification of patients at high risk of chronicity. Quantitative and qualitative analysis of cytokines, chemokines, and oxidative stress parameters provides valuable insights into the underlying molecular mechanisms, offering a promising foundation for personalized patient management.

Therapeutic interventions must be comprehensive and tailored to the biochemical and immunopathogenetic characteristics of each case. The rational use of anti-inflammatory agents, targeted antimicrobial therapy that considers the presence of microbial biofilms, antioxidant supplementation, and correction of allergic sensitization allows for directed modulation of the principal pathogenetic pathways, minimizing the risk of chronic disease and improving clinical outcomes. In selected cases, surgical interventions such as functional endoscopic sinus surgery or adenoidectomy are indicated to restore sinus drainage and ventilation, further reducing inflammatory burden and enhancing recovery.

In conclusion, the integration of clinical, biochemical, and immunopathogenetic approaches constitutes a modern, evidence-based framework for the diagnosis and management of pediatric rhinosinusitis. This integrated strategy not only facilitates the development of individualized treatment plans but also holds the potential to improve quality of life, reduce recurrence rates, and prevent long-term complications in affected children. Future research focusing on elucidating molecular inflammatory pathways and identifying novel biochemical markers may enable the creation of targeted therapeutic interventions, advancing precision medicine in pediatric otorhinolaryngology.

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