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CHRONIC RHINOSINUSITIS: PATHOPHYSIOLOGY, DIAGNOSIS, AND EVIDENCE-BASED TREATMENT

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Annotation

The article discusses current concepts of chronic inflammation of the paranasal sinuses in children (chronic rhinosinusitis, CRS) based on the European Position Paper on Rhinosinusitis and Nasal Polyps 2012 (EPOS 2012). The structure of bacterial pathogens, trigger factors of CRS, and modern treatment recommendations are presented. Special attention is given to the use of topical antibacterial therapy, particularly fusafungine (Bioparox), in children over 12 years of age with CRS.

Keywords:

chronic rhinosinusitis, children, topical antibacterial therapy, fusafungine, EPOS, nasal polyps.

Article Text

Chronic rhinosinusitis (CRS) remains an urgent problem in pediatrics and otorhinolaryngology due to the persistent increase in incidence. According to various authors, some form of rhinosinusitis affects 5–15% of adults and approximately 5% of children [1].

The EPOS 2012 guidelines define pediatric rhinosinusitis as inflammation of the nasal and paranasal sinus mucosa, characterized by two or more symptoms, one of which must be nasal congestion/obstruction or nasal discharge (anterior or posterior), and may also include facial pain or cough. Additionally, diagnostic criteria include endoscopic findings (nasal polyps, mucopurulent discharge predominantly from the

middle meatus, mucosal edema) and CT evidence of mucosal changes in the ostiomeatal complex or paranasal sinuses [2,3].

In pediatric practice, CRS is typically a multifactorial condition. Unlike acute rhinosinusitis, the etiology of chronic inflammation in children often involves non-infectious factors. CRS may be a manifestation of systemic conditions such as primary and secondary immunodeficiencies, disorders associated with abnormal mucus viscosity (cystic fibrosis), and diseases involving impaired ciliary function (Kartagener syndrome, primary ciliary dyskinesia) [2,4]. The role of gastroesophageal reflux in CRS is also being actively discussed.

Based on etiological factors, CRS may be bacterial, fungal, or caused by bacterial–fungal associations. Depending on severity, it may be classified as mild, moderate, or severe. Morphologically, CRS can be catarrhal, purulent, polypoid–purulent, or polypoid. Despite intensive research, the etiology and pathogenesis of nasal polyps remain incompletely understood [4]. Recent data highlight the significance of allergic mechanisms, arachidonic acid metabolism disorders, and persistence of microbial superantigens. In children, inherited disorders—especially cystic fibrosis—are of particular relevance. Around 37% of adults with cystic fibrosis have nasal polyps [5], while pediatric data suggest rates of 39.1% [6]. Some national studies report up to 84.6% among children with mixed or respiratory forms of cystic fibrosis [7]. Overall, 15–20% of children with polypoid CRS have cystic fibrosis [6,7].

Multiple pathological conditions may predispose children to CRS by impairing aeration and mucociliary clearance of the paranasal sinuses. These include anatomical anomalies of the nasal cavity and sinuses, chronic rhinitis, atopy, NSAID intolerance, immunodeficiency, disorders of mucociliary transport, gastroesophageal reflux disease, and orosinusal fistulas [8,9].

The modern functional concept of CRS pathogenesis is based on the principle that chronic inflammation is predominantly secondary to obstructed ventilation and

drainage of the paranasal sinuses [10]. Even minimal mucosal edema due to inflammation may disrupt mucociliary transport within narrow sinus outlets. Anatomical variants in the ostiomeatal complex, as well as septal spurs and crests, may further obstruct natural sinus ostia, resulting in hypoxia, secretion stasis, and prolonged retention of pathogens.

The role of microorganisms in CRS, once considered definitive, is now actively re-evaluated. Studies show that bacterial cultures from the middle meatus often reveal aerobic (86%) and anaerobic (8–10%) pathogens. Among aerobes, *Staphylococcus aureus*, coagulase-negative staphylococci, and *Streptococcus pneumoniae* predominate. Atypical intracellular pathogens (chlamydia, mycoplasma) may persist in epithelial and lymphoid tissues, contributing to severe disease. Standard culture methods do not detect these organisms, requiring serology and PCR diagnostics.

Ineffective antimicrobial therapy against intracellular pathogens may contribute to disease chronicity.

Another important factor in CRS pathogenesis is the formation of bacterial biofilms—complex microbial communities embedded in an extracellular matrix. Biofilm-associated bacteria exhibit significantly increased resistance to antibiotics, sometimes up to 1000-fold. Known biofilm-forming species implicated in CRS include *Staphylococcus spp.*, *Streptococcus spp.*, *H. influenzae*, *M. catarrhalis*, and *P. aeruginosa*. Biofilms have been identified in 80–100% of sinus mucosa biopsies in CRS patients.

The emergence of antibiotic-resistant strains complicates systemic therapy. The goals of antimicrobial treatment include symptom control, eradication of pathogens, and prevention of chronic mucosal damage. However, irrational or prolonged antibiotic use promotes resistance [11]. EPOS provides clear criteria for CRS diagnosis and treatment. Recent research has focused on topical antibacterial therapies.

One such agent is fusafungine (Bioparox), a topical antibiotic with anti-inflammatory properties. Its aerosol particles (<1 μm) ensure deep penetration into sinonasal mucosa. Fusafungine inhibits interleukin-1, TNF, and free radicals while preserving phagocytosis. It is active against gram-positive and gram-negative bacteria, including methicillin-resistant staphylococci, *H. influenzae*, *Legionella*, *Mycoplasma pneumoniae*, and *Candida albicans*, reducing the risk of secondary candidiasis.

Randomized trials conducted by national researchers have confirmed its efficacy in various forms of rhinosinusitis [12]. Comprehensive evaluations—including nasal endoscopy, mucociliary clearance assessment, thermometry, rhinomanometry, and cytology—have shown faster restoration of mucosal function in children over 12 years receiving fusafungine as part of therapy [13]. The use of topical antibiotics may reduce unnecessary systemic antibiotic use and limit resistance development.

Based on current evidence, the etiology and pathogenesis of CRS remain incompletely understood, underscoring the need for further research. Despite available guidelines, additional studies are essential to improve clinical outcomes.

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