Evidence Based Review of Sinusitis Classification Pathophysiology Diagnosis and Management

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ABSTRACT

Sinusitis, more accurately termed rhinosinusitis because inflammation of the nasal cavity and paranasal sinuses usually coexists, is one of the most frequent upper airway disorders encountered in primary care, pediatrics, allergy, and otorhinolaryngology. It is traditionally classified by duration into acute rhinosinusitis, recurrent acute rhinosinusitis, and chronic rhinosinusitis, and by phenotype into disease with or without nasal polyps. Although most acute episodes are viral and self-limiting, a smaller proportion progress to acute bacterial rhinosinusitis, where diagnostic accuracy is essential to avoid unnecessary antibiotic exposure and antimicrobial resistance. Chronic rhinosinusitis is a heterogeneous inflammatory syndrome lasting at least 12 weeks and requiring objective evidence of mucosal disease on nasal endoscopy or computed tomography. Current evidence indicates that chronic disease is not simply a persistent infection; rather, it reflects complex interactions among epithelial barrier dysfunction, impaired mucociliary clearance, microbial dysbiosis, type 1, type 2, or type 3 immune pathways, allergy, asthma, aspirin-exacerbated respiratory disease, environmental exposures, and host genetic susceptibility. This review summarizes contemporary evidence on sinusitis epidemiology, classification, pathophysiology, diagnostic work-up, and treatment. A narrative review methodology was used to synthesize international guidelines, systematic reviews, randomized trials, and high-impact observational studies published mainly between 2004 and 2026. Evidence-based management emphasizes symptomatic treatment, saline irrigation, intranasal corticosteroids, careful selection of antibiotics in probable bacterial disease, short courses of systemic corticosteroids in selected chronic rhinosinusitis with nasal polyps, endoscopic sinus surgery for medically refractory disease, and biologic therapies for severe uncontrolled type 2 chronic rhinosinusitis with nasal polyps.

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Introduction

Sinusitis is a common but often imprecisely used clinical term. In modern rhinology, the preferred term is rhinosinusitis, because inflammation of the paranasal sinus mucosa almost always occurs together with inflammation of the nasal mucosa. The disease spectrum includes acute, recurrent, and chronic forms, with different mechanisms, diagnostic requirements, and treatment priorities. Contemporary guidelines from Europe, North America, and international rhinology societies emphasize that a diagnosis should not be made on facial pain alone, because nasal obstruction, nasal discharge, smell impairment, and objective inflammatory findings are central to case definition [1,2,3].

Acute rhinosinusitis (ARS) usually follows viral upper respiratory tract infection and lasts less than 4 weeks. Most cases are viral and improve without antibiotics. Acute bacterial rhinosinusitis (ABRS) is suspected when symptoms persist beyond approximately 10 days without improvement, are severe at onset, or worsen after initial improvement, often described as “double worsening” [4,5]. This distinction is clinically important because antibiotic benefit is modest in uncomplicated acute disease, while adverse effects and antimicrobial resistance remain substantial public health concerns [14,15].

Recurrent acute rhinosinusitis is usually defined as multiple discrete ABRS episodes per year with complete or near-complete resolution between episodes. It differs from chronic rhinosinusitis because persistent baseline inflammation is absent between attacks. Recurrent disease should prompt assessment for allergic rhinitis, anatomic obstruction, immune deficiency, ciliary dysfunction, environmental exposure, and odontogenic sources when clinically indicated [1,3].

Chronic rhinosinusitis (CRS) is defined by symptoms lasting at least 12 weeks, usually including nasal obstruction and/or nasal discharge, with additional

symptoms such as facial pressure, hyposmia, or anosmia, and with objective evidence of sinonasal inflammation on nasal endoscopy or computed tomography [1,2,3]. CRS is commonly divided into CRS without nasal polyps (CRSsNP) and CRS with nasal polyps (CRSwNP). This phenotype-based division remains clinically useful because CRSwNP is more strongly associated with type 2 inflammation, asthma, aspirin-exacerbated respiratory disease, smell loss, recurrence after surgery, and biologic eligibility [7,23,27].

The burden of sinusitis is considerable. It affects quality of life, sleep, smell, work productivity, school performance, and healthcare utilization. CRS can impair disease-specific quality of life to a degree comparable with other chronic diseases, especially when associated with asthma, severe nasal obstruction, anosmia, recurrent infections, or repeated systemic corticosteroid use [7,12]. In clinical practice, sinusitis is also a frequent driver of antibiotic prescribing. Therefore, the central challenge is not only to treat symptoms but also to separate viral self-limited disease from bacterial disease, and inflammatory chronic disease from infection-dominant conditions [4,9,14].

Modern understanding has moved beyond the older view that sinusitis is mainly retained infected mucus caused by blocked sinus ostia. Ostial obstruction, impaired ventilation, and microbial factors remain relevant, but CRS is now better conceptualized as a chronic inflammatory disorder of the unified upper airway. The sinonasal epithelium is an active immune organ, producing cytokines, chemokines, antimicrobial peptides, and barrier proteins. When epithelial barrier function and mucociliary clearance are disrupted, environmental irritants, microbes, allergens, and host immune pathways can perpetuate inflammation [10,11,22].

The clinical importance of this shift is substantial. If sinusitis is treated only as infection, antibiotics will be overused and inflammatory drivers will remain uncontrolled. Conversely, if bacterial complications or odontogenic infection are missed, delayed treatment may lead to orbital or intracranial complications. A balanced approach requires classification by duration, severity, objective findings, phenotype, endotype, comorbidities, and previous treatment response [1,3,4].

Materials and methods

This article was prepared as a narrative review of sinusitis and rhinosinusitis, focusing on clinically relevant evidence for classification, pathophysiology, diagnosis, and management. The review question was: What is the current evidence-based approach to the diagnosis and treatment of acute and chronic rhinosinusitis in children and adults?

A structured literature search strategy was developed using PubMed, Google Scholar, guideline repositories, and journal databases. Search terms included: "acute rhinosinusitis," "acute bacterial rhinosinusitis," "chronic rhinosinusitis," "chronic rhinosinusitis with nasal polyps," "sinusitis guideline," "EPOS 2020," "ICAR rhinosinusitis," "IDSA acute bacterial rhinosinusitis," "adult sinusitis guideline," "pediatric sinusitis," "nasal polyps biologics," "dupilumab chronic rhinosinusitis," "mepolizumab nasal polyps," "omalizumab nasal polyps," "endoscopic sinus surgery chronic rhinosinusitis," "SNOT-22," "Lund-Mackay score," "nasal microbiome chronic rhinosinusitis," and "type 2 inflammation chronic rhinosinusitis."

Priority was given to international guidelines, systematic reviews, meta-analyses, Cochrane reviews, randomized controlled trials, large observational studies, and high-impact mechanistic reviews. Key guideline sources included the European Position Paper on Rhinosinusitis and Nasal Polyps 2020, the International Consensus Statement on Allergy and Rhinology: Rhinosinusitis 2021, the Infectious Diseases Society of America guideline for ABRS, the American Academy of Otolaryngology–Head and Neck Surgery adult sinusitis guideline, and the American Academy of Pediatrics guideline for pediatric ABRS [1,2,3,4,5,6]. Foundational studies on patient-reported outcomes, computed tomography scoring, antibiotic effectiveness, topical therapy, systemic corticosteroids, surgery, microbiome, endotypes, and biologics were also included [12,13,14,17,18,19,20,21,24,25,26].

The inclusion criteria were: articles in English; studies or guidelines addressing human sinusitis/rhinosinusitis; publication in peer-reviewed journals or recognized clinical guideline platforms; relevance to diagnosis, classification, mechanisms, or therapy; and traceability through PubMed or Google Scholar. Seminal older studies were retained when they introduced widely used diagnostic tools or mechanistic concepts, such as the Lund-Mackay computed tomography score and the SNOT-22 quality-of-life instrument [12,13].

The exclusion criteria were: isolated case reports unless they described major complications; non-peer-reviewed opinion pieces; articles focused only on rare fungal, malignant, or skull-base diseases unless relevant to differential diagnosis; and studies not

clearly applicable to clinical sinusitis care. Because this was a narrative rather than a formal systematic review, no pooled quantitative meta-analysis was performed. Instead, the evidence was synthesized thematically across six domains: classification, epidemiology, pathophysiology, diagnosis, medical therapy, and procedural or biologic therapy.

Reference numbering follows the order in which sources are first cited in the manuscript. In-text citations are presented in numerical bracket style, as requested, while the reference list is formatted in APA 7 style with numbering added for ease of citation matching.

Epidemiology, Risk Factors, and Disease Burden

Rhinosinusitis is highly prevalent, but reported rates vary depending on diagnostic criteria. Symptom-based surveys often overestimate CRS because nasal obstruction, discharge, facial pressure, and smell disturbance overlap with allergic rhinitis, nonallergic rhinitis, migraine, viral infection, dental disease, and other conditions. When objective evidence is required, prevalence estimates are lower but clinically more specific [7,8]. This distinction is important because guideline-defined CRS requires symptoms plus objective inflammation, not symptoms alone [1,2,3].

Acute viral rhinosinusitis is extremely common because it often occurs during viral upper respiratory tract infection. A minority of patients develop ABRS. The exact proportion varies by population and diagnostic criteria, but most uncomplicated acute episodes improve spontaneously. This explains why randomized trials and meta-analyses show only modest average benefit from antibiotics in clinically diagnosed acute rhinosinusitis [14,15]. The clinical implication is that watchful waiting, symptomatic care, and safety-net instructions are appropriate for many uncomplicated cases, while antibiotics should be reserved for persistent, severe, or worsening presentations consistent with ABRS [1,4,5].

CRS has a more complex epidemiology. Risk factors include asthma, allergic rhinitis, aspirin-exacerbated respiratory disease, smoking, air pollution, occupational irritants, immune deficiency, cystic fibrosis, primary ciliary dyskinesia, dental infection, anatomic variation, and prior sinonasal surgery [7,8,9]. Some of these are causal contributors, whereas others are associated comorbidities that modify symptoms, severity, or treatment response. For example, asthma and CRS frequently coexist within the "united airway" concept, and CRSwNP with asthma often reflects type 2 inflammation across both upper and lower airways [7,23].

Disease burden is multidimensional. Patients commonly report nasal blockage, anterior or posterior rhinorrhea, facial pressure, reduced smell, fatigue, poor sleep, impaired concentration, and reduced productivity. The SNOT-22 questionnaire is widely used to measure disease-specific health-related quality of life and is sensitive to clinically meaningful change after medical or surgical intervention [12]. Objective findings, such as CT opacification, may not always correlate closely with symptom severity; therefore, patient-reported outcomes are essential for assessing disease impact and treatment response [12,13].

The economic burden includes physician visits, imaging, medications, antibiotics, systemic corticosteroid courses, surgery, absenteeism, presenteeism, and treatment of comorbid asthma or allergy. Inappropriate antibiotic prescribing adds further societal cost through antimicrobial resistance and adverse drug events. Repeated systemic corticosteroid courses, often used in severe CRSwNP, can improve symptoms transiently but expose patients to cumulative risks such as hyperglycemia, hypertension, mood disturbance, insomnia, osteoporosis, cataracts, and adrenal suppression [18,19]. These risks partly explain the growing role of biologic therapies in severe uncontrolled type 2 CRSwNP [24,25,26].

Sinusitis burden is also affected by under-recognition of specific subtypes. Odontogenic sinusitis, for instance, is commonly unilateral and may present with foul smell, dental symptoms, or unilateral maxillary disease on imaging. Allergic fungal rhinosinusitis is often associated with nasal polyps, allergic mucin, fungal sensitization, and characteristic CT findings. Immunodeficiency-related disease may present with recurrent bacterial infections or poor response to standard therapy. These subgroups require targeted evaluation rather than repeated empirical treatment [1,3].

In children, sinusitis must be interpreted in the context of developing sinus anatomy, frequent viral infections, adenoid disease, allergic rhinitis, and environmental exposures. Pediatric ABRS is diagnosed clinically, and routine imaging is not recommended for uncomplicated cases because imaging abnormalities are common after viral infection and do not reliably

distinguish viral from bacterial disease [6]. However, orbital swelling, severe headache, neurologic signs, toxic appearance, or suspected complications require urgent evaluation and imaging [6].

Overall, the epidemiology of sinusitis supports a stratified approach. The most common acute cases require conservative management; probable bacterial cases require selective antibiotics; chronic inflammatory disease requires objective confirmation and long-term anti-inflammatory therapy; and severe refractory CRSwNP requires phenotype- and endotype-directed escalation.

Pathophysiology and Phenotypes

The pathophysiology of sinusitis depends on whether the disease is acute or chronic. Acute viral rhinosinusitis begins with viral infection of the upper airway mucosa, leading to edema, increased mucus production, temporary obstruction of sinus drainage pathways, and impaired mucociliary clearance. Secondary bacterial infection may develop when mucus stasis and mucosal inflammation create favorable conditions for bacterial overgrowth. Common ABRS pathogens include Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis, and, in selected contexts, Staphylococcus aureus and anaerobes, especially with odontogenic sources [4,6,9].

CRS is not simply prolonged ABRS. It is better understood as a heterogeneous inflammatory disorder involving epithelial barrier dysfunction, innate and adaptive immune activation, tissue remodeling, mucociliary impairment, neurogenic inflammation, microbial dysbiosis, and environmental exposure [10,11]. The sinonasal epithelium acts as a physical barrier and immune signaling interface. When epithelial tight junctions, antimicrobial peptides, mucin regulation, and ciliary function are impaired, the mucosa becomes more vulnerable to allergens, pollutants, viruses, bacteria, and fungi [10,11].

Historically, CRS was classified into CRSsNP and CRSwNP. This phenotype remains useful but incomplete. Current research increasingly emphasizes endotypes, meaning biologically distinct inflammatory pathways. Type 2 inflammation is characterized by cytokines such as interleukin-4, interleukin-5, and interleukin-13, eosinophilia, local IgE production, mast cell activation, and tissue remodeling. It is strongly associated with CRSwNP, asthma, aspirin-exacerbated respiratory disease, smell loss, recurrence, and response to biologics targeting type 2 pathways [23,24,25,26,27].

Non-type 2 CRS includes type 1 and type 3 inflammatory patterns, often involving interferon-gamma, interleukin-17, neutrophilic inflammation, and different microbial or epithelial signatures. Geographic differences are important: type 2 CRSwNP predominates in many Western cohorts, whereas mixed and non-type 2 patterns have historically been more frequently described in parts of Asia, although these patterns are changing over time [11,23,27]. This heterogeneity explains why a single treatment pathway is insufficient for all CRS patients.

The microbiome plays a modifying role. Older infection-centered models focused on culturable bacteria, but culture-independent sequencing has shown that sinonasal microbial communities are complex. CRS may involve dysbiosis rather than infection by a single organism. Staphylococcus aureus is particularly important in CRSwNP because colonization and enterotoxin-specific IgE responses may amplify type 2 inflammation and local immune activation [21,22]. However, the presence of bacteria does not automatically mean antibiotics will be effective, because microbes may be bystanders, modifiers, or components of biofilm communities embedded within chronic inflammation [20,21].

Biofilms have been detected in CRS and may contribute to persistence, antibiotic tolerance, and postoperative recurrence. Yet biofilm detection is not routinely used in standard practice because methods are not widely standardized and treatment implications remain uncertain. Similarly, fungi can be present in the sinonasal cavity without causing invasive disease. Allergic fungal rhinosinusitis is a distinct inflammatory subtype and should not be confused with ordinary fungal colonization [1,3].

Mucociliary clearance is another central mechanism. Ciliary dysfunction may be primary, as in primary ciliary dyskinesia, or secondary to inflammation, infection, smoking, pollution, dehydration, or surgery. Impaired clearance allows mucus retention and prolongs contact between the mucosa and inflammatory stimuli. Saline irrigation is biologically plausible because it improves mucus clearance, removes crusts and inflammatory mediators, and improves topical drug delivery, although clinical effect varies by technique, volume, adherence, and disease phenotype [17].

Comorbidities shape disease expression. Allergic rhinitis can mimic or exacerbate CRS symptoms and

should be treated when present. Asthma and CRS can aggravate each other, particularly in CRSwNP. Aspirin-exacerbated respiratory disease is characterized by asthma, nasal polyps, and respiratory reactions to cyclooxygenase-1 inhibitors; it is often associated with severe recurrent polyposis and may require specialized management, including leukotriene modifiers, aspirin desensitization in selected patients, surgery, or biologics [1,3,23].

Therefore, sinusitis pathophysiology should be interpreted through a layered model: acute infection and mucosal edema dominate many short-duration cases; chronic inflammatory immune pathways dominate CRS; and microbial, allergic, anatomic, dental, genetic, and environmental factors modify severity and recurrence.

Diagnosis and Differentiation

The diagnosis of sinusitis begins with careful history. Essential features include duration, pattern, severity, nasal obstruction, anterior or posterior discharge, facial pain or pressure, fever, smell dysfunction, cough, dental symptoms, allergic triggers, asthma, aspirin sensitivity, immune status, prior antibiotic use, prior surgery, and red flags. Time course is crucial. Symptoms lasting less than 10 days and gradually improving usually suggest viral rhinosinusitis. Persistent symptoms beyond 10 days, severe onset with high fever and purulent discharge, or double worsening support ABRS [4,5].

Physical examination should include anterior rhinoscopy when possible. Findings may include mucosal edema, purulent drainage, polyps, crusting, septal deviation, turbinate hypertrophy, and signs of allergic rhinitis. However, primary care diagnosis of acute disease is often clinical. Routine imaging is not recommended for uncomplicated acute rhinosinusitis because CT abnormalities are common during viral infections and cannot reliably distinguish viral from bacterial inflammation [4,5,6].

Nasal endoscopy is valuable in recurrent, chronic, unilateral, severe, atypical, or refractory disease. It can identify middle meatal purulence, polyps, edema, crusting, fungal debris, masses, cerebrospinal fluid leak suspicion, and postoperative anatomy. Endoscopy also allows targeted culture in selected patients, which is preferable to non-directed nasal swabs when culture is needed. Culture may be considered for immunocompromised patients, treatment failures, severe disease, unusual organisms, or suspected complications [1,3].

For CRS, objective confirmation is required. Computed tomography of the paranasal sinuses is the imaging modality of choice when symptoms persist despite appropriate therapy, when surgery is considered, or when an alternative diagnosis must be excluded [1,3]. The Lund-Mackay score remains widely used in research and clinical communication; it grades sinus opacification and ostiomeatal complex obstruction on CT [13]. However, CT severity does not perfectly correlate with symptoms, so imaging should complement rather than replace clinical assessment and patient-reported outcomes [12,13].

Magnetic resonance imaging is not first-line for routine CRS but is useful when soft tissue characterization is needed, such as suspected tumor, invasive fungal disease, intracranial extension, orbital complication, or encephalocele. Dental evaluation is important when imaging shows unilateral maxillary sinusitis, periapical disease, oroantral fistula, dental implant complications, or foul-smelling unilateral discharge. Odontogenic sinusitis often requires dental source control in addition to sinonasal management [1,3].

Laboratory testing is not routinely necessary for uncomplicated acute disease. In CRS, testing should be individualized. Allergy testing is appropriate when symptoms suggest allergic rhinitis or when allergy control may improve outcomes. Immune evaluation may be indicated in recurrent bacterial infections, severe refractory disease, unusual organisms, or poor vaccine responses. Evaluation for cystic fibrosis or primary ciliary dyskinesia is appropriate in selected children and adults with early-onset disease, bronchiectasis, chronic otitis media, infertility, neonatal respiratory distress, or situs abnormalities [1,3,6].

The differential diagnosis is broad. Migraine and tension-type headache are frequently misdiagnosed as "sinus headache," especially when facial pressure occurs without purulent discharge or objective inflammation. Allergic rhinitis causes congestion, rhinorrhea, sneezing, itching, and postnasal drip but does not necessarily indicate sinus disease. Nonallergic rhinitis, medication-induced rhinitis, gastroesophageal reflux, dental disease, temporomandibular joint disorder, trigeminal neuralgia, neoplasm, granulomatosis with polyangiitis, sarcoidosis, and cerebrospinal fluid leak may mimic sinusitis [1,3,9].

Red flags require urgent assessment. Periorbital edema, proptosis, ophthalmoplegia, reduced visual acuity, severe frontal headache, altered mental status, meningismus, focal neurologic deficits, persistent high fever, forehead swelling, or toxic appearance may indicate orbital cellulitis, subperiosteal abscess, cavernous sinus thrombosis, meningitis, brain abscess, or osteomyelitis [4,6]. These situations require urgent imaging, specialist involvement, and intravenous therapy.

Patient-reported outcome measures should be integrated into CRS care. SNOT-22 is especially useful for baseline severity, shared decision-making, and treatment monitoring [12]. Smell testing, asthma control tools, endoscopic polyp scoring, CT scores, and medication burden can further define severity. The best diagnostic approach combines symptoms, objective findings, phenotype, comorbidities, and response to previous therapy.

Evidence-Based Management

Management depends on whether disease is acute viral, probable bacterial, recurrent acute, chronic without polyps, or chronic with polyps. In acute viral rhinosinusitis, treatment is supportive. Analgesics, antipyretics, hydration, saline sprays or irrigation, and selected short-term decongestants may reduce symptoms. Intranasal corticosteroids may help when allergic rhinitis or significant nasal inflammation is present, but routine antibiotics are not indicated for viral disease [1,4,5].

For uncomplicated ABRS, guidelines support either watchful waiting with reliable follow-up or antibiotics, depending on severity, duration, patient preference, comorbidity, and risk of complications [1,4,5]. Antibiotic selection should consider local resistance, allergy history, recent antibiotic exposure, and severity. Amoxicillin-clavulanate is commonly recommended as first-line therapy in many guidelines, while respiratory fluoroquinolones should generally be reserved because of safety concerns and antimicrobial stewardship principles [4,5]. Meta-analyses show that antibiotics provide only small average benefit in many adults with clinically diagnosed acute rhinosinusitis, reinforcing the need for careful selection [14,15].

Pediatric ABRS is diagnosed clinically using persistent, worsening, or severe symptom patterns. Antibiotics are recommended for severe onset or worsening disease, while observation may be reasonable for selected persistent but non-severe cases with appropriate follow-up [6]. Imaging is avoided in uncomplicated pediatric cases but is essential when orbital or intracranial complications are suspected [6].

CRS management begins with education, trigger control, and long-term topical therapy. High-volume saline irrigation is commonly recommended because it is safe, inexpensive, and mechanistically useful for mucus clearance and topical drug distribution [17]. Intranasal corticosteroids are foundational therapy, especially in CRSwNP, and reduce mucosal inflammation with a favorable safety profile compared with systemic corticosteroids [18]. Delivery technique matters: sprays may not reach the sinuses well in obstructed or unoperated anatomy, while high-volume irrigations or steroid irrigations are often used after surgery under specialist supervision [1,3].

Systemic antibiotics have a limited role in CRS. They may be appropriate for acute bacterial exacerbations, culture-directed infection, or selected cases with purulence, but evidence does not support repeated empirical antibiotics for all CRS patients [20]. Long-term macrolide therapy has been investigated because of possible anti-inflammatory effects, but benefits appear inconsistent and may depend on phenotype, endotype, IgE status, and population. Risks include gastrointestinal effects, QT prolongation, drug interactions, and antimicrobial resistance [20].

Short courses of systemic corticosteroids can rapidly reduce polyp size and improve obstruction and smell in CRSwNP, but benefits are often temporary and cumulative adverse effects are clinically important [19]. Therefore, systemic corticosteroids should be used selectively, at the lowest effective exposure, and with caution in patients with diabetes, hypertension, osteoporosis, glaucoma, infection risk, psychiatric vulnerability, or pregnancy. Repeated steroid dependence should prompt reassessment and escalation planning [1,3,19].

Endoscopic sinus surgery is indicated for medically refractory CRS, recurrent complications, obstructive polyposis, fungal disease, mucoceles, selected odontogenic cases, or when access is needed for topical therapy. Surgery is not simply "removal of infection"; it improves ventilation, drainage, inflammatory tissue burden, and delivery of topical medications. Prospective studies suggest that appropriately selected patients with refractory CRS and impaired quality of life often improve more after endoscopic sinus surgery than with continued medical therapy alone [28,29]. However, surgery does not cure

the underlying inflammatory tendency, especially in severe CRSwNP, so postoperative topical therapy and long-term follow-up remain essential [1,3].

Biologic therapies have changed the management of severe uncontrolled CRSwNP. Dupilumab, targeting interleukin-4 and interleukin-13 signaling, improved polyp size, sinus opacification, smell, congestion, and quality-of-life outcomes in phase 3 trials [24]. Omalizumab, targeting IgE, improved endoscopic and patient-reported outcomes in severe nasal polyposis [25]. Mepolizumab, targeting interleukin-5, improved nasal polyp size and obstruction in recurrent refractory severe CRSwNP [26]. Benralizumab, targeting the interleukin-5 receptor alpha and depleting eosinophils, has also shown significant improvements in polyp and blockage outcomes, although biologic choice should be individualized [30].

Biologics are generally considered when CRSwNP remains uncontrolled despite appropriate intranasal corticosteroids, saline irrigation, and usually prior surgery or systemic corticosteroid need, especially when type 2 features are present. Selection should consider asthma, blood eosinophils, total IgE, allergy, aspirin-exacerbated respiratory disease, smell loss, prior surgery, systemic steroid burden, patient preference, cost, access, dosing schedule, and expected response domains [1,3,24,25,26,30]. Response should be reassessed using symptoms, SNOT-22, smell, endoscopic polyp score, systemic steroid use, asthma control, and need for surgery.

Future directions include biomarker-guided care, improved endotype classification, microbiome-informed therapy, better drug delivery systems, artificial intelligence for imaging and outcome prediction, and comparative effectiveness studies of surgery versus biologics and combined strategies. Current biomarkers are helpful but imperfect. Blood eosinophils, tissue eosinophilia, total IgE, periostin, fractional exhaled nitric oxide, and cytokine signatures may support type 2 classification, but no single biomarker fully predicts treatment response [11,23,27].

A practical stepwise approach is recommended. First, classify by duration and severity. Second, confirm CRS objectively. Third, identify phenotype: CRSsNP, CRSwNP, unilateral disease, odontogenic disease, allergic fungal disease, or systemic disease. Fourth, treat modifiable drivers such as allergic rhinitis, asthma, smoking, occupational exposure, dental infection, and medication contributors. Fifth, use topical anti-inflammatory therapy consistently. Sixth, escalate to surgery or biologics for refractory disease based on objective findings, quality-of-life burden, inflammatory pattern, comorbidities, and patient goals [1,3].

Conclusions

In conclusion, sinusitis is not a single disease but a spectrum of acute infectious and chronic inflammatory disorders. The strongest contemporary approach is diagnostic precision followed by targeted, evidence-based therapy. Antibiotics should be used judiciously, topical therapies should be optimized, surgery should be reserved for appropriate refractory or structural disease, and biologics should be selected for severe uncontrolled type 2 CRSwNP. This integrated approach can reduce overtreatment, improve quality of life, and align sinusitis care with modern precision medicine.

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