

Pediatric Allergic Rhinitis



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KEYWORDS

- Allergic rhinitis • Conjunctivitis • Concurrent conditions • Avoidance
- Pharmacotherapy • Immunomodulation

KEY POINTS

- Allergic rhinitis significantly affects sleep, cognition, performance, and quality of life.
- Allergic rhinitis is diagnosed clinically based on compatible history and risk factors, characteristic signs and symptoms, and the confirmed presence of allergen-specific immunoglobulin E (if clinically indicated).
- For most patients, allergic rhinitis is a persistent condition that requires years of ongoing management, which combines allergen avoidance strategies and pharmacotherapy, with consideration of allergen immunotherapy for severe or refractory cases.
- Treatment of allergic rhinitis improves concurrent asthma, and allergen immunotherapy might potentially prevent asthma in some children with allergic rhinitis.
- Optimized partnership between patient, caregiver, and health care professional helps to maximize response to allergic rhinitis treatment, while reducing/allaying concerns about safety or therapeutic complications.

INTRODUCTION

Allergic rhinitis (AR) is a chronic immunoglobulin (Ig)E-dependent respiratory disease of the upper airway characterized by rhinorrhea, sneezing, congestion, and/or nasocular pruritus.¹ Estimates for its prevalence vary regionally and by age group. Several risk factors have been proposed or identified (**Box 1**). The International Study of Asthma and Allergies in Childhood systematically evaluated the prevalence of asthma, allergic rhinoconjunctivitis, and eczema in approximately 1.2 million children in 98 countries and determined the overall prevalence of AR in children aged 6 to 7 years and 13 to 14 years was 8.5% and 14.6%, respectively.²

CLINICAL MANIFESTATIONS

AR characteristically presents with paroxysms of rhinorrhea, sneezing, and congestion, often accompanied by pruritus of the eyes, nose, and palate. Postnasal drainage,

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Box 1**Risk factors for pediatric allergic rhinitis**

Family history

Male gender

Firstborn status

Early systemic use of antibiotics

Maternal smoking

Exposure to indoor aeroallergens

Serum IgE greater than 100 IU/mL before age 6 years

Presence of allergen-specific IgE

Data from Wallace DV, Dykewicz MS, Bernstein DI, et al. The diagnosis and management of rhinitis: an updated practice parameter. *J Allergy Clin Immunol* 2008;122:S1–84; Matheson MC, Dharmage SC, Abramson MJ, et al. Early-life risk factors and incidence of rhinitis: results from the European Community Respiratory Health Study—an international population-based cohort study. *J Allergy Clin Immunol* 2011;128:816–23; Saulyte J, Regueira C, Montes-Martinez A, et al. Active or passive exposure to tobacco smoking and allergic rhinitis, allergic dermatitis, and food allergy in adults and children: a systematic review and meta-analysis. *PLoS Med* 2014;11:e1001611.

cough, fatigue, and irritability are other common symptoms.^{1,3,4} Of these, nasal congestion is the most common.⁵ Young children, however, typically do not blow their noses, but they may instead exhibit repeated sniffing, snorting, throat-clearing, and coughing. Additionally, a palatal click can sometimes be audible if children use their tongues to scratch their itchy palates. Chronic mouth breathing owing to nasal obstruction in children with AR has been linked to facial abnormalities and an increased incidence of dental malocclusions and snoring.^{6,7}

COGNITIVE FUNCTION AND QUALITY OF LIFE

Sleep-disordered breathing, fatigue, and generalized malaise are important sequelae of undertreated AR.^{5,8,9} Irritability and behavioral issues are often seen as a result of AR in children.^{9,10} Children with AR are more likely to have emotional and social complications.⁵

AR can also impair cognitive functioning and academic performance.^{5,11,12} Sedating antihistamines can be unintentional cofactors.¹³ Complications of AR such as sleep disorders and eustachian tube dysfunction with associated conductive hearing loss likely contribute to learning impairment.¹⁴

CONCURRENT CONDITIONS

AR occurs in association with several other diseases, including asthma, allergic conjunctivitis, and sinusitis.

Asthma

Asthma and rhinitis often coexist and are believed to represent a spectrum of the same disease entity. Rhinitis is correlated with and is a risk factor for the occurrence and severity of asthma. Up to 50% of individuals with chronic rhinitis have asthma. Conversely, most individuals with asthma also have chronic rhinitis.¹⁵ Accordingly, expert panels recommend that patients with AR should be evaluated for asthma and vice versa, and, if identified, the 2 conditions should be treated concurrently.^{15,16}

Allergic Conjunctivitis

Up to 60% of patients with AR have concurrent conjunctivitis.¹⁷ Pediatric data are similar. For example, Ibáñez and colleagues¹⁸ reported 53.6% of children with AR had ocular symptoms and found a correlation between severity and duration of AR and the likelihood of concurrent conjunctivitis. Allergic conjunctivitis characteristically presents with bilateral pruritus, an urge to rub the eyes, tearing (watery discharge), hyperemia and chemosis, and conjunctival edema. Eyelid edema is also common.

Sinusitis

Nasal inflammation from AR can obstruct the sinus ostiomeatal complex, contributing to acute or chronic sinusitis. Symptoms of bacterial sinusitis include nasal congestion, mucopurulent drainage, facial/dental discomfort, and cough. Mucopurulent drainage and cough are the most useful predictors of bacterial sinusitis in children, but no single symptom has high predictive value for discriminating between bacterial sinusitis and rhinitis.¹⁹

Oral Allergy Syndrome (Pollen-Food Allergy Syndrome)

Oral allergy syndrome (OAS) is a localized form of food allergy that develops in some pollen-allergic individuals who ingest immunologically related, uncooked fruits (eg, apples, peaches, cucumbers, melons) or raw vegetables during the pertinent pollen season. Characteristic symptoms include pruritus, tingling, redness, and swelling of the lips, tongue, and soft palate. OAS symptoms have been reported in 25% of children with AR.²⁰ Sensitization to birch, the most common pollen sensitization reported with OAS, was found in 31% of children with AR who ingested fruits (eg, apples) sharing birch pollen-related proteins.²⁰

Other Conditions

AR probably contributes to eustachian tube dysfunction, causing concurrent otitis media with effusion, which can be associated with conductive hearing loss and language delay.^{21,22} Nasal obstruction caused by severe AR can also cause sleep-disordered breathing and anosmia.^{23,24}

PATTERNS OF SYMPTOMS

AR has traditionally been classified as either seasonal or perennial, depending on whether symptoms occur at a particular time of year or are present throughout the year. The US Food and Drug Administration continues to use this classification in its approval process for AR therapeutics. The Allergic Rhinitis in Asthma guidelines developed in collaboration with the World Health Organization classify AR according to temporal pattern (intermittent or persistent) and symptomatic severity (mild or moderate/severe) (**Box 2**).

DIAGNOSIS

AR is diagnosed clinically based on compatible history and risk factors, characteristic signs and symptoms, and the confirmed presence of allergen-specific IgE (if clinically indicated).

HISTORY

Seasonal/intermittent AR is often diagnosed by history alone, because each recurring pollen season produces symptoms. For example, symptoms caused by tree and

Box 2**Allergic Rhinitis in Asthma classification of allergic rhinitis**

Intermittent means that the symptoms are present:

- Less than 4 d/wk
- Or for less than 4 weeks

Persistent means that the symptoms are present:

- More than 4 d/wk
- And for more than 4 weeks

Mild means that none of the following items are present:

- Sleep disturbance
- Impairment of daily activities, leisure, and/or sport
- Impairment of school or work
- Troublesome symptoms

Moderate-severe means that one or more of the following items are present:

- Sleep disturbance
- Impairment of daily activities, leisure, and/or sport
- Impairment of school or work
- Troublesome symptoms

From Bousquet J, Van Cauwenberge P, Khaltaev N, et al. Allergic rhinitis and its impact on asthma. J Allergy Clin Immunol 2001;108:S147–334; with permission.

grass pollen allergy typically occur in the spring, whereas those caused by ragweed pollen occur in the fall, although exposures may vary regionally. Clinical history alone might also permit the diagnosis of episodic AR if there is an obvious, consistent association between exposure and symptom onset (eg, sneezing commences within minutes of visiting with an individual who has indoor cats). In contrast, the culprit allergens (eg, dust mites or animal emanations) in perennial/persistent AR might not be evident by history alone.

PHYSICAL FINDINGS

The nose, oropharynx, tympanic membranes, and eyes should each be examined for findings of AR or concurrent conditions. The nasal mucosa in active disease often has mucosal edema accompanied by pallor or a pale bluish hue. Clear drainage may be visible anteriorly or posteriorly. Posterior drainage is often observed with hyperplastic lymphoid tissue lining the posterior pharynx (cobblestoning). An effusion of serous fluid may accumulate behind the tympanic membrane when significant nasal mucosal edema and eustachian tube dysfunction are present.

Certain facial features, when present, also suggest AR.²⁵ A transverse nasal crease can be caused by a child's repeated vertical rubbing of the tip of the nose (allergic salute). Infraorbital edema and discoloration caused by venous stasis is often described as allergic shiners. Dennie-Morgan lines are accentuated folds below the lower eyelids that suggest allergic conjunctivitis. Allergic facies, observed in some young children, consist of chronic mouth breathing, dental malocclusion, and high-arched palate.

NATURAL HISTORY

AR is uncommon in children younger than 2 years, because it typically requires a few years of allergen exposure to develop.²⁶ If a very young child has persistent nasal symptoms, another diagnosis should be considered (see Differential Diagnosis in later discussion). In children, sensitization (presence of allergen-specific IgE) precedes the appearance of clinical allergy, which develop first to allergens continuously present in the respirable environment (eg, dust mites, animal emanations) and then to pollens and other seasonal aeroallergens.²⁰ In a prospective study of 2024 children, AR prevalence increased from 5% at age 4 years to 14% at age 8 years.²⁰ Up to 80% of patients with AR have symptoms before age 20 years.²⁷

DIAGNOSTIC TESTING

Diagnostic testing is not necessary before making the presumptive diagnosis of AR and initiating empiric treatment. However, allergen-specific IgE testing can confirm the diagnosis and facilitate individualized AR management. Care should be taken to select for testing only those allergens considered clinically relevant based on the history, clinical manifestations, and local aerobiology, as clinically irrelevant results can occur.²⁸ Skin prick testing (SPT) and in vitro specific-IgE (sIgE) testing are sensitive diagnostic methods. Properly performed SPT is generally preferred because it is less expensive than sIgE testing and provides rapid results, which the patient/caregiver can observe.¹ SPT is usually performed by allergy/immunology specialists because rare systemic reactions during testing are possible. Conversely, sIgE testing might be preferable for children with extensive dermatitis, persistent need for medication that interferes with the SPT wheal-and-flare response, or a relative contraindication to SPT (eg, uncontrolled asthma).¹

DIFFERENTIAL DIAGNOSIS

Various disorders can mimic AR or coexist with it. **Box 3** provides an age-based differential diagnosis for pediatric AR.

PATHOPHYSIOLOGY

Sensitized individuals respond to allergen exposure by producing allergen-specific IgE. IgE antibodies bind to IgE receptors on mast cells throughout the respiratory mucosa and to circulating basophils. When the same allergen is subsequently inhaled, it binds to IgE molecules, which cross-link on the mast cell surface, resulting in activation and release of inflammatory mediators. Nasal mast cells release histamine, leukotrienes, prostaglandins, bradykinin, and platelet-activating factor, among other mediators, which produce the clinical stigmata of AR. Tissue eosinophilia is also a feature of AR, and eosinophilic mediators augment nasal inflammation. The allergic nasal response consists of an immediate phase, which peaks 15 to 30 minutes after allergen exposure and corresponds to mast cell mediator release, followed in approximately 60% to 70% of individuals by a late phase, which peaks 6 to 12 hours after exposure and corresponds to infiltration of nasal mucosa with inflammatory cells (eg, eosinophils, basophils, T lymphocytes).^{29,30}

MANAGEMENT STRATEGIES

Optimized partnership between patient, caregiver, and health care professional helps to maximize response to AR treatment while reducing/allaying concerns about safety

Box 3

Age-based differential diagnosis for pediatric allergic rhinitis

Infants (0–1 y)

- Infectious rhinitis: viral URI, sinusitis, chlamydia, syphilis
- Allergic rhinitis: indoor and food allergens
- Nonallergic, noninfectious rhinitis
 - Irritant rhinitis
- Obstruction
 - Anatomic abnormality: trisomy 21
 - Partial or unilateral choanal atresia
 - Tumor: encephalocele, meningocele

Toddlers (1–3 y)

- Allergic rhinitis: indoor and food allergens
- Infectious rhinitis: recurrent URI, sinusitis (CF, primary ciliary dyskinesia, immunodeficiency^a)
- Nonallergic, noninfectious rhinitis
 - Irritant rhinitis
- Obstruction
 - Adenoidal or turbinate hypertrophy
 - Foreign body

Children (3–12 y)

- Allergic rhinitis: indoor and outdoor allergens
- Infectious rhinitis: recurrent URI, sinusitis (CF, primary ciliary dyskinesia, immunodeficiency^a)
- Nonallergic, noninfectious rhinitis
 - Physical rhinitis (gustatory, cold, sunlight)
 - Irritant rhinitis
 - NARES
 - Rhinitis medicamentosa: topical decongestants
- Obstruction
 - Adenoidal or turbinate hypertrophy
 - Foreign body
 - Nasal polyp
 - Tumor

Adolescents (12–18 y)

- Allergic rhinitis: indoor and outdoor allergens
- Infectious rhinitis: viral URI, sinusitis (immunodeficiency^a)
- Nonallergic, noninfectious rhinitis
 - Physical rhinitis (gustatory, cold, sunlight)
 - NARES
 - Rhinitis medicamentosa: topical decongestants, cocaine
 - Idiopathic (vasomotor)
- Obstruction
 - Adenoidal or turbinate hypertrophy
 - Nasal polyps, septal deviation
 - Tumor: angiofibroma

Abbreviations: CF, cystic fibrosis; NARES, nonallergic rhinitis with eosinophilia syndrome; URI, upper respiratory infection.

^a IgA deficiency, hypogammaglobulinemia, human immunodeficiency virus infection.

Adapted from Zacharisen MC. Rhinitis in children, adolescents, the elderly, and pregnant women: special considerations. *Immunol Allergy Clin North Am* 2000;20:428; with permission.

or therapeutic complications. The 4 key components of AR management are patient/caregiver education, environmental control measures, pharmacotherapy, and immunomodulation.

Environmental Control Measures

Environmental control by avoidance of relevant allergens and symptomatic triggers is a key initial step in the management of AR. However, complete avoidance is rarely possible. Evidence on pet allergen avoidance is lacking, and recent reviews have found minimal to no evidence to support dust mite avoidance measures, the most common of which are impermeable pillow and mattress covers.^{31,32} Avoidance of respiratory irritants, such as tobacco smoke and strong perfumes, seems reasonable.

Pharmacotherapy

Because of the limited effectiveness of environmental control measures, pharmacotherapeutics are often needed for children with moderate/severe or persistent AR symptoms. **Fig. 1** summarizes guideline-directed therapy, which may involve more than one agent.^{1,7,15} Treatment of AR with intranasal corticosteroids, antihistamines, or leukotriene modifiers is also effective in reducing or controlling asthma symptoms.¹⁵

Corticosteroids

Systemic corticosteroids should rarely be used in children with AR because safer, effective alternatives are available.^{1,7,15} Intranasal corticosteroids (INCS) are the most effective therapy for AR in children.^{1,15,33} INCSs treat the inflammatory clinical manifestations of AR and have also shown great benefit in improving the comorbidities of asthma and bronchial hyperresponsiveness, conjunctivitis, sleep disorders, and quality-of-life impairment.^{34–38}

There is no evidence to indicate that any INCS is clinically superior if used correctly at the recommended dose.³⁹ Symptomatic improvement has been reported within 12 hours of use.⁴⁰ In the Pediatric Allergies in America survey, 19% of parents reported that INCSs provided relief for their child in less than 1 hour and 31% in 1 hour.⁵ However, full benefit may not be achieved for days to weeks after starting therapy.

The potential for systemic absorption of INCSs to have effects on the hypothalamic-pituitary-adrenal axis and on growth has been evaluated in many studies, most of which showed limited or no hypothalamic-pituitary-adrenal effects at recommended doses.^{41–45} Second-generation INCSs have markedly lower bioavailability compared with first-generation agents, resulting in a lower risk of side effects (**Table 1**). Longer-term studies of the growth effects caused by inhaled corticosteroids administered for asthma are also reassuring. Caution is needed, however, if corticosteroids are used for concurrent conditions, as the adverse effects of topical corticosteroid preparations can be additive. Appropriate recommendations for children prescribed INCSs include (1) lowest effective dose, (2) regular height measurements, and (3) periodic re-evaluation of need for INCS.

Oral and Intranasal Antihistamines

Second-generation oral and intranasal antihistamines (**Table 2**) are effective for AR and are generally well tolerated.^{1,15} Metabolites of second-generation antihistamines, such as fexofenadine (the metabolite of terfenadine) or levocetirizine (a purified isomer of cetirizine), were designed to have fewer central nervous system effects, although

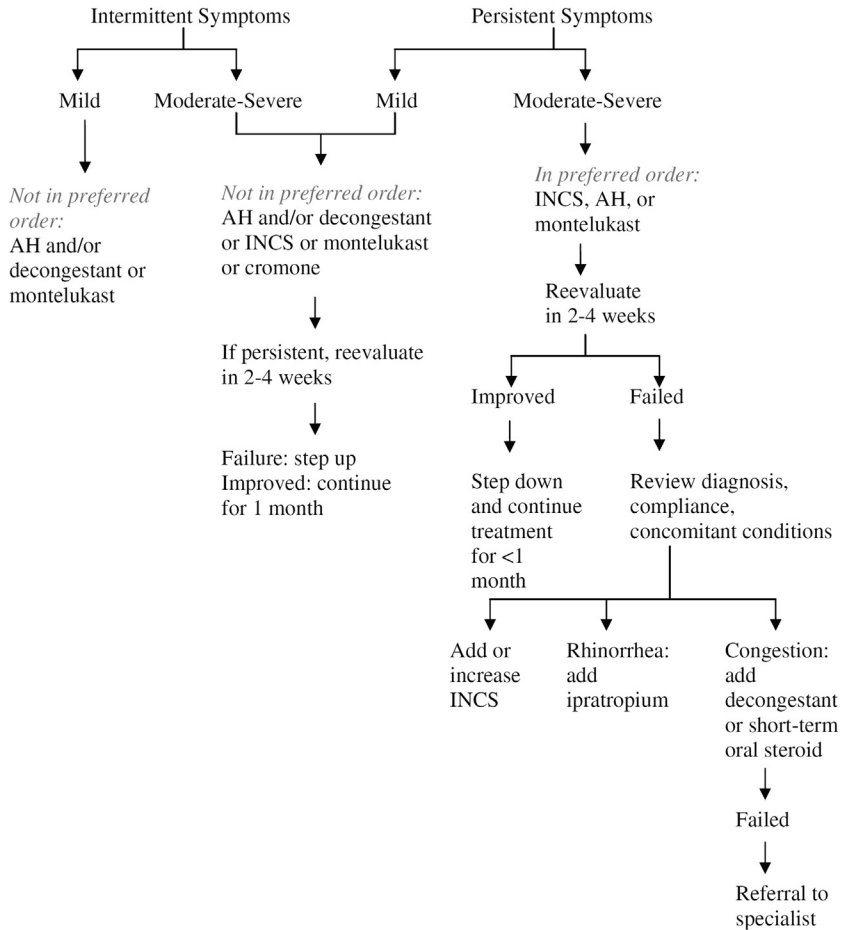


Fig. 1. Suggested treatment algorithm for pediatric allergic rhinitis. Consider allergen avoidance measures, intranasal antihistamines, and immunomodulatory therapy where appropriate. AH, antihistamines. (From Gentile D, Bartholow A, Valovirta E, et al. Current and future directions in pediatric allergic rhinitis. *J Allergy Clin Immunol: In Practice* 2013;1:218; with permission.)

this has not been confirmed.⁴⁶ Antihistamine sprays have a rapid onset of action (less than 15 minutes after use).⁴⁰ Dual azelastine-fluticasone therapy is also available.

First-generation antihistamines should no longer be used in children because of their risk of sedation and other unfavorable effects.¹³

Leukotriene Modifiers

Montelukast has been effective in seasonal and perennial AR pediatric studies.^{47,48} It is the only leukotriene modifier approved in the United States for the treatment of pediatric AR. Adverse neuropsychiatric changes have been reported in some individuals, but studies have shown no significant difference when compared with placebo.^{49,50} Pending further information, professional societies have advised that patients might continue montelukast unless mood disturbance develops.⁵¹

Table 1
Intranasal preparations used in pediatric AR

Generic Name	Brand Name(s)	Aqueous/ Aerosol	MOA	RX/OTC	Dosing
Azelastine	Astelin	Aqueous	AH	RX	5–11 y: 1 SEN BID; ≥ 12 y: 2 SEN BID (137 $\mu\text{g/spray}$)
Azelastine	Astepro	Aqueous	AH	RX	6–11 y: 1 SEN BID; ≥ 12 y: 1–2 SEN BID (205.5 $\mu\text{g/spray}$)
Azelastine/Fluticasone propionate	Dymista	Aqueous	AH/CS	RX	≥ 12 y: 1 SEN BID (137 $\mu\text{g}/50$ $\mu\text{g/spray}$)
Beclomethasone dipropionate	Beconase AQ	Aqueous	CS	RX	≥ 6 y: 1–2 SEN BID (42 $\mu\text{g/spray}$)
Beclomethasone dipropionate	Qnasl	Aerosol	CS	RX	≥ 12 y: 2 SEN daily (80 $\mu\text{g/spray}$)
Budesonide	Rhinocort Aqua	Aqueous	CS	RX	≥ 6 y: 1–2 SEN daily (32 $\mu\text{g/spray}$)
Ciclesonide	Omnanis	Aqueous	CS	RX	≥ 6 y: 2 SEN daily (50 $\mu\text{g/spray}$)
Ciclesonide	Zetonna	Aerosol	CS	RX	≥ 12 y: 1 SEN daily (37 mcg/spray)
Flunisolide	(Brand name product no longer available in US)	Aqueous	CS	RX	6–14 y: 2 SEN BID; ≥ 15 y: 2 SEN BID-TID (max 8 SEN per day) (25,29 $\mu\text{g/spray}$)
Cromolyn	NasalCrom	Aqueous	Chromone	OTC	≥ 2 y: 1 SEN TID-QID (max 6 SEN per day)
Fluticasone furoate	Veramyst	Aqueous	CS	RX	≥ 2 y: 1–2 SEN daily (27.5 $\mu\text{g/spray}$)
Fluticasone propionate	Flonase	Aqueous	CS	RX	≥ 4 y: 1–2 SEN daily (50 $\mu\text{g/spray}$)
Ipratropium bromide	Atrovent	Aqueous	AC	RX	≥ 6 y: 2 SEN BID-TID (42 μg ; 0.03% spray)
Mometasone furoate	Nasonex	Aqueous	CS	RX	2–11 y: 1 SEN daily; ≥ 12 y: 2 SEN daily (50 $\mu\text{g/spray}$)
Olopatadine	Patanase	Aqueous	AH	RX	6–11 y: 1 SEN BID; ≥ 12 y: 2 SEN BID (665 $\mu\text{g/spray}$)
Oxymetazoline	Afrin	Aqueous	Topical Vasoconstrictor	OTC	≥ 6 y: 1–2 SEN BID for up to 3 d
Triamcinolone	Nasacort AQ	Aqueous	CS	OTC	2–5 y: 1 SEN daily; ≥ 6 y: 1–2 SEN daily (55 $\mu\text{g/spray}$)

Fluticasone propionate, fluticasone furoate, mometasone furoate, and ciclesonide are second-generation intranasal CS with undetectable to less than 2% systemic bioavailability. The remaining intranasal CS are first-generation agents, which have 10%–50% bioavailability.

Abbreviations: AC, anticholinergic; AH, antihistamine; BID, twice daily; CS, corticosteroid; MOA, mechanism of action; OTC, over the counter; QD, once daily; QID, four times daily; RX, prescription; SEN, spray each nostril; TID, three times daily.

Data from deShazo RD, Kemp SF. Pharmacotherapy of allergic rhinitis. In: Post TW, editor. UpToDate. Waltham (MA): UpToDate. Accessed May 10, 2014.

Table 2
Oral preparations used in pediatric AR

Generic Name	Brand Name	MOA	RX/OTC	Dosing
Cetirizine	Zyrtec	AH	OTC	6 mo–1 y: 2.5 mg daily; 1–2 y: 2.5 mg daily or BID; 2–5 y: 2.5–5 mg daily; ≥6 y: 5–10 mg daily
Desloratadine	Clarinet	AH	RX	6–11 mo: 1 mg daily; 1–5 y: 1.25 mg daily; 6–11 y: 2.5 mg daily; ≥12 y: 5 mg daily
Fexofenadine	Allegra	AH	OTC	2–11 y: 30 mg BID; ≥12 y: 60 mg BID or 180 mg daily
Levocetirizine	Xyzal	AH	RX	6 mo–5 y: 1.25 mg QPM; 6–11 y: 2.5 mg QPM; ≥12 y: 2.5–5 mg QPM
Loratadine	Alavert; Claritin	AH	OTC	2–6 y: 5 mg daily; ≥6 y: 10 mg daily
Montelukast	Singulair	LTRA	RX	6–23 mo: 4 mg (oral granules) daily; 2–5 y: 4 mg (tablet or granules) daily; 6–14 y: 5 mg daily; ≥15 y: 10 mg daily

Abbreviations: AH, antihistamine; BID, twice daily; LTRA, leukotriene receptor antagonist; MOA, mechanism of action; OTC, over the counter; QPM, once nightly; RX, prescription; SEN, spray each nostril; TID, three times daily.

Intranasal Cromolyn

Cromolyn sodium is a weakly effective topical mast cell stabilizer that is well tolerated but needs to be administered 4 times daily for efficacy.¹⁵ It can also be used prophylactically for episodic rhinitis before allergen exposure.

Intranasal Anticholinergics

Ipratropium nasal spray can limit nasal drainage but offers no relief for pruritus or congestion. It is sometimes useful in children with profuse rhinorrhea uncontrolled by INCS use.¹

Intranasal Decongestants

Vasoconstrictor decongestant sprays, such as oxymetazoline, can be used briefly in children to relieve severe nasal congestion, but prolonged use can lead to rhinitis medicamentosa characterized by rebound nasal congestion.¹ In contrast, the combination of intranasal decongestant and INCS in adults may effectively treat nasal congestion without rhinitis medicamentosa,⁵² but this approach has not been evaluated in children.

Saline Irrigation

As adjunctive therapy, nasal irrigation with saline prepared from distilled or sterilized water may assist AR management by clearing secretions, allergens, and irritants.^{53,54} Irrigation devices should be cleaned as directed and replaced regularly.

IMMUNOMODULATION

Allergen Immunotherapy

Allergen immunotherapy (AIT) encompasses both subcutaneous immunotherapy (SCIT) and sublingual immunotherapy. SCIT involves the supervised administration of gradually increasing doses of therapeutic vaccines of aeroallergens to which an individual has shown allergen-specific IgE. Over time, an optimal maintenance dose is

attained that effectively modulates the immune response to the aeroallergen(s), resulting in fewer symptoms on exposure. Multiple immunomodulatory mechanisms are probably involved.⁵⁵ AIT is unique among therapeutic modalities in its ability to provide sustained immunomodulation of allergic respiratory disease, including the reduced incidence of asthma in children with AR.^{56,57}

Sublingual immunotherapy involves the application of allergen (typically one allergen only) to the sublingual tissues or oral mucosa either as oral solutions or rapidly disintegrating tablets. Compared with SCIT, it has significantly lower risk and little difference in overall efficacy based on meta-analyses.^{58,59} Two grass preparations and one ragweed preparation received US Food and Drug Administration approval for use in the United States in 2014.

Anti-immunoglobulin E Therapy

Omalizumab is a recombinant human monoclonal IgG anti-IgE antibody that binds to circulating IgE to prevent its binding to the surface receptors of mast cells and basophils. For patients 12 years and older, anti-IgE is effective for moderate-to-severe persistent asthma and for concurrent AR.^{7,15}

REFERRAL

An allergist/immunologist can be particularly helpful for children with any of the following:

- Severe or prolonged symptoms despite avoidance measures and pharmacotherapy; AIT has been found to alter the progression of allergic respiratory disease, including the prevention of subsequent asthma development.
- Comorbid conditions
- Intolerance of pharmacotherapy
- Interest in immunomodulation as a therapeutic option

SUMMARY

AR presents both challenges and opportunities for the pediatric patient, caregiver, and health care professional. Proper diagnosis facilitates patient/caregiver education and permits consideration of multiple therapeutic modalities tailored to the individual patient. AR management in a specific child is age dependent and influenced by the severity and frequency of the symptoms and the presence of any concurrent conditions. Current strategies permit symptomatic control and improved quality of life for most patients.

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