Allergic rhinitis: Definition, epidemiology, pathophysiology, detection, and diagnosis

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Allergic rhinitis (AR) is a heterogeneous disorder that despite its high prevalence is often undiagnosed. It is characterized by one or more symptoms including sneezing, itching, nasal congestion, and rhinorrhea. Many causative agents have been linked to AR including pollens, molds, dust mites, and animal dander. Seasonal allergic rhinitis (SAR) is fairly easy to identify because of the rapid and reproducible onset and offset of symptoms in association with pollen exposure. Perennial AR is often more difficult to detect than SAR because of the overlap with sinusitis, respiratory infections, and vasomotor rhinitis. SAR can result in hyperresponsiveness to allergens such as cigarette smoke, once pollen season is over. Perennial AR is defined as occurring during approximately 9 months of the year. AR affects an estimated 20 to 40 million people in the United States alone, and the incidence is increasing; an estimated 20% of cases are SAR; 40% of cases are perennial rhinitis; and 40% of cases are mixed. The pathophysiology of SAR is complex. There is a strong genetic component to the allergic response, which is driven through mucosal infiltration and action on plasma cells, mast cells, and eosinophils. The allergic response occurs in two phases, which are considered the "early" and "late" phase responses. Early phase response occurs within minutes of exposure to the allergen and tends to produce sneezing, itching, and clear rhinorrhea; late phase response occurs 4 to 8 hours after allergen exposure and is characterized by congestion, fatigue, malaise, irritability, and possibly neurocognitive deficits. The key to diagnosis of AR is awareness of signs and symptoms. IgE antibody tests to detect specific allergens are the standard method used today; however, in addition, diagnosis must be confirmed with a positive history and demonstration that the symptoms are the result of IgE-mediated inflammation. (J Allergy Clin Immunol 2001;108:S2-8.)

Key words: *Allergic rhinitis, IgE, mast cells, perennial rhinitis*

Rhinitis is a heterogeneous disorder characterized by one or more of the following nasal symptoms: sneezing, itching, rhinorrhea, and/or nasal congestion. Rhinitis frequently is accompanied by symptoms involving the eyes, ears, and throat, including postnasal drainage.

Abbreviations used

AR: Allergic rhinitis
IL: Interleukin
IgE: Gamma globulin E
LT: Leukotriene
PAR: Perennial allergic rhinitis
SAR: Seasonal allergic rhinitis
T_H2: T helper lymphocyte 2

There are many different causes of rhinitis in children and adults. Approximately 50% of all cases of rhinitis are caused by allergy. In the case of rhinitis caused by allergens, symptoms arise as a result of inflammation induced by a gamma globulin E (IgE)-mediated immune response to specific allergens such as pollens, molds, animal dander, and dust mites. The immune response involves the release of inflammatory mediators and the activation and recruitment of cells to the nasal mucosa.[1,2] The various causes of rhinitis, which make up the differential diagnosis for AR, are listed in Table I.

EPIDEMIOLOGY

Although allergic rhinitis (AR) reportedly occurs very frequently, data regarding the true underlying causes of rhinitis are difficult to interpret. Most population surveys rely on physician-diagnosed rhinitis for their data, possibly underestimating the actual frequency with which rhinitis occurs. Some population studies have been conducted by means of questionnaires administered to subjects, followed by telephone interviews to attempt to make a specific diagnosis of rhinitis. Results of such studies reflect a more accurate prevalence of rhinitis but are likely to continue to underreport this disease.[2-12]

Most epidemiologic studies have been directed toward the identification of seasonal allergic rhinitis (SAR), or hay fever, because of the easy identification of the rapid and reproducible onset and offset of symptoms in association with pollen exposure. Perennial allergic rhinitis (PAR) is more difficult to identify because its symptom complex often overlaps with chronic sinusitis, recurrent upper respiratory infections, and vasomotor rhinitis.

The reported prevalence of rhinitis in epidemiologic studies, conducted in various countries, ranges from 3% to 19%. Studies that have included the most information would suggest that SAR (hay fever) is found in approximately 10% of the general population and perennial rhinitis in 10% to 20% of the population.[2-12] Overall, allergic rhinitis affects 20 to 40 million people in the United States.[11,12]

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The frequency of AR in the general population appears to be increasing. Swedish army studies have shown that the prevalence of hay fever has increased from 4% to 8% in the 10 years from 1971 to 1981.[13] Additionally, atopic skin test reactivity increased from 39% to 50% in Tucson, Arizona, during an 8-year period of testing.[5,12]

The prevalence of AR in the pediatric population also appears to be rising. One study showed a prevalence of physician-diagnosed AR in 42% of 6-year-old children.[5] One recent study conducted in Finland[14] reported a near tripling of the prevalence from 1977 through 1979, to 1991. Currently, AR is the most common allergic disease and one of the leading chronic conditions in children <18 years of age.[15]

Sex

In childhood, boys with AR outnumber girls, but, in general, equal numbers are affected during adulthood.

Age

Symptoms of AR develop before the age of 20 years in 80% of cases. Children in families with a bilateral family history of allergy generally have symptoms before puberty; those with a unilateral family history tend to have symptoms later in life or not at all.[5-7] Symptoms of AR develop in 1 of 5 children by 2 to 3 years of age and in approximately 40% by age 6 years. Approximately 30% develop symptoms during adolescence.

Risk factors

Studies have shown that the frequency of AR increases with age and that positive allergy skin tests are significant risk factors for the development of new symptoms of hay fever. There appears to be a higher prevalence of rhinitis in higher socioeconomic classes, in nonwhites, in some polluted areas, in individuals with a family history of allergy, and in individuals born during the pollen season. Additionally, AR is more likely to occur in firstborn children. Studies in children in the first years of life have shown that the risk of rhinitis was higher in those youngsters with early introduction of foods or formula, heavy maternal cigarette smoking in the first year of life, exposure to indoor allergens such as animal dander and dust mites, higher serum IgE levels (>100 IU/mL before age 6), the presence of positive allergen skin-prick tests, and parental allergic disorders.[5]

Social/economic impact

Because of the high prevalence of AR, impaired quality of life, costs of treatment, and presence of comorbidities such as asthma, sinusitis, and otitis media, AR has a tremendous impact on society. The severity of AR ranges from mild to seriously debilitating. The cost of treating AR and indirect costs related to loss of workplace productivity resulting from the disease are significant and substantial. The estimated cost of AR, based on direct and indirect costs, is 2.7 billion dollars for the year 1995, exclusive of costs for associated medical problems such as sinusitis and asthma.[16] In children with AR, the quality of life of both the parents and the child, including the ability to learn,[17] is affected.

PATHOPHYSIOLOGY

Under normal conditions, the nasal mucosa quite efficiently humidifies and cleans inspired air. This is the result of orchestrated interactions of local and humoral mediators of host defense.[18] In AR, these mechanisms go awry and contribute to the signs and symptoms of the disorder.[19]

Components of the allergic response

The allergic sensitization that characterizes AR has a strong genetic component. The tendency to develop IgE/mast cell/T_H2 lymphocyte immune responses is inherited by atopic patients. Exposure to threshold concentrations of dust mite fecal proteins; cockroach allergen; cat, dog, and other danders; pollen grains; or other allergens for prolonged periods of time leads to the presentation of the allergen by antigen presenting cells to CD4+ T lymphocytes, which then release interleukin (IL)-3, IL-4, IL-5, and other T_H2 cytokines. These cytokines drive proinflammatory processes, such as IgE production, against these allergens through the mucosal infiltration and actions of plasma cells, mast cells, and eosinophils.

Once the patient has become sensitized to allergens, subsequent exposures trigger a cascade of events that result in the symptoms of AR. The allergic response in AR can be divided into two phases, the immediate or early phase response and the late-phase response.

Early phase

During periods of continuous allergen exposure, increasing numbers of IgE-coated mast cells traverse the epithelium, recognize the mucosally deposited allergen, and degranulate.[20] Products of this degranulation include preformed mediators such as histamine, tryptase (mast-cell specific marker), chymase ("connective tissue" mast cells only), kininogenase (generates bradykinin), heparin, and other enzymes. In addition, mast cells secrete several inflammatory mediators de novo (ie, not preformed and stored in mast cell granules) including prostaglandin D_2 and the sulfidopeptidyl leukotrienes (LT)C_4, LTD_4, and LTE_4. These mediators cause blood vessels to leak and produce the mucosal edema plus watery rhinorrhea characteristic of AR. Glands secrete mucoglycoconjugates and antimicrobial compounds and dilate blood vessels to cause sinusoidal filling and thus occlusion and congestion of nasal air passages. These mediators also stimulate sensory nerves, which convey the sensations of nasal itch and congestion, and recruit systemic reflexes such as sneezing. The above responses develop within minutes of allergen exposure and are thus termed the early phase, or "immediate," allergic response.[21] Sneezing, itching, and copious clear rhinorrhea are characteristic symptoms during early phase allergic responses, although some degree of nasal congestion also can occur.

Late phase

The mast cell–derived mediators released during early phase responses are hypothesized to act on postcapillary endothelial cells to promote the expression of vascular cell adhesion molecule and E-selectin, which facilitate the adhesion of circulating leukocytes to the endothelial cells. Chemoattractant cytokines such as IL-5 promote the infiltration of the mucosa with eosinophils, neutrophils, and basophils; T lymphocytes; and macrophages.[22,23] During the 4- to 8-hour period after allergen exposure, these cells become activated and release inflammatory mediators, which in turn reactivate many of the proinflammatory reactions of the immediate response. This cellular-driven late inflammatory reaction is termed the "late phase response." This reaction may be clinically indistinguishable from the immediate reaction, but congestion tends to predominate.[24] Eosinophil-derived mediators such as major basic protein, eosinophil cationic protein, and leukotrienes have been shown to damage the epithelium, leading ultimately to the clinical and histologic pictures of chronic allergic disease.

Subsets of the T-helper lymphocytes are the likely orchestrators of the chronic inflammatory response to allergens. T_H2 lymphocytes promote the allergic response by releasing IL-3, IL-4, IL-5, and other cytokines that promote IgE production, eosinophil chemoattraction and survival in tissues, and mast cell recruitment.[25] Cytokines released from T_H2 lymphocytes and other cells may circulate to the hypothalamus and result in the fatigue, malaise, irritability, and neurocognitive deficits that commonly are noted in patients with AR. Cytokines produced during late phase allergic responses can be reduced by glucocorticoids.[26]

When subjects are challenged intranasally with allergen repeatedly, the amount of allergen required to produce an immediate response decreases.[27] This effect is termed "priming" and is hypothesized to be a result of the influx of inflammatory cells that occurs during late phase allergic responses. The response is clinically significant because exposure to an allergen may promote an exaggerated response to other allergens. Priming highlights the importance of fully defining the spectrum of allergens for a given patient and the need to prevent this process by initiating preseasonal, prophylactic, anti-inflammatory therapy.

Classification of allergic rhinitis

On the basis of timing and duration of allergen exposure, and thus the allergen pathogeneses, AR is classified as seasonal or perennial. Overall, approximately 20% of all cases are strictly seasonal, 40% perennial, and 40% mixed (perennial with seasonal exacerbations).

Seasonal allergic rhinitis

Tree, grass, and weed pollens and outdoor mold spores are common seasonal allergens. The symptoms typically appear during a defined season in which aeroallergens are abundant in the outdoor air. The length of seasonal exposure to these allergens is dependent on geographic location. Therefore, familiarity with the pollinating season of the major trees, grasses, and weeds of the locale makes the syndrome easier to diagnose.[28] Certain outdoor mold spores also display seasonal variation, with highest levels in the summer and fall months.[29]

Typical symptoms during pollen exposure include the explosive onset of profuse watery rhinorrhea, itching, and sneezing, along with frequent allergic symptoms of the eye. Congestion also occurs but usually is not the most troubling symptom. The onset and offset of symptoms usually track the seasonal pollen counts. However, hyperresponsiveness to irritant triggers, which develops from the inflammatory reaction of the late phase and priming responses, often persists after cessation of the pollen season. Such triggers include tobacco smoke, noxious odors, changes in temperature, and exercise.

Perennial allergic rhinitis

Year-round exposure to dust mites, cockroaches, indoor molds, and cat, dog, and other danders leads to persistent tissue edema and infiltration with eosinophils, mast cells, T_H2 lymphocytes, and macrophages.[30] PAR can also be caused by pollen in areas where pollen is prevalent perennially.

A universally accepted definition of perennial rhinitis does not exist. Most often, it is defined as a disease that persists for longer than 9 months each year and produces two or more of the following symptoms: serous or seromucus hypersecretion, nasal blockage caused by a swollen nasal mucosa, or sneezing paroxysms. Nasal congestion and mucous production (postnasal drip) symptoms predominate in most patients, and sneezing, itching, and watery rhinorrhea may be minimal.

Perennial allergic rhinitis with seasonal exacerbations

Symptoms of AR may also be perennial with seasonal exacerbation, depending on the spectrum of allergen sensitivities.

DETECTION/DIAGNOSIS

Detection

A careful history and physical examination are the most effective diagnostic maneuvers for the identification of AR in children.[31] The key to accurate and timely diagnosis in children is a heightened awareness of the condition and its potential comorbidities. AR in children is often undiagnosed or misdiagnosed as other disorders such as recurrent colds. When cough is predominant, especially at night, AR may be misdiagnosed as "cough-variant" asthma. To make a correct diagnosis with appropriate accuracy and timeliness, the clinician must be knowledgeable and attentive of the symptoms and signs of rhinitis, ask specific questions directed at the presence and cause of rhinitis symptoms at each well-child visit, and understand the differential diagnosis of AR in children[2,11] (see Table I). One must be aware of the comorbidities of AR (asthma, sinusitis, otitis

media), pursue specific diagnostic testing when indicated, and often administer therapeutic trials of anti-inflammatory medications.

Parents must also be aware of signs and symptoms and report them to physicians, because the more subtle case presentations may otherwise go undiagnosed. Such underdiagnosis may be responsible for substantial morbidity in children, who often do not report their symptoms. Unfortunately, children who live with allergic symptoms on a daily basis for prolonged periods of time may mistakenly assume that their altered state is normal.

Typical symptoms of AR include sneezing, itching, clear rhinorrhea, and congestion. Congestion may be bilateral or unilateral or may alternate from side to side. It is generally more pronounced at night. With nasal obstruction, the patient is likely to be a mouth-breather, and snoring can be a nocturnal symptom. As such, sleep disturbances may indicate the presence of an allergic disorder. With chronic disease, abnormalities of facial development, dental malocclusion, and the allergic facies may ensue, with an open mouth and gaping habitus.

Whereas older children blow their noses frequently, younger children do not. Instead, they sniff, snort, and repetitively clear their throats. Nasal pruritus may stimulate grimacing and twitching and picking of the nose. The latter may result in epistaxis. Children often have the allergic salute, an upward rubbing of the nose with the palm of the hand. This often produces an allergic nasal crease, an accentuated, horizontal skin fold over the lower third of the nose. Children with AR also may have recurrent sinusitis or otitis media, eczema, or asthma.

Patients may also complain of red, itchy eyes, along with itchy throat and ears. They may also lose their senses of smell and taste. Increased symptoms are frequently noted with increased exposure to the responsible allergen, such as after cutting grass or sleeping on a feather pillow.

With development of the allergic reaction, clear nasal secretions will be evident, and the nasal mucous membranes will become edematous without much erythema. The mucosa appears boggy and blue-gray. With continued exposure to the allergen, the turbinates will appear swollen and can obstruct the nasal airway. Conjunctival edema, itching, tearing, and hyperemia are frequent findings in patients with associated allergic conjunctivitis. AR patients, particularly children with significant nasal obstruction and venous congestion, may also demonstrate edema and darkening of the tissues beneath the eyes. These so-called "shiners" are not pathognomonic for AR because they also can be seen in patients with chronic rhinitis and/or sinusitis. Thick, purulent nasal secretions generally indicate the presence of infection, including the possibility of sinusitis.

In severe cases, especially during the peak pollen season, mucous membranes of the eyes, eustachian tube, middle ear, and paranasal sinuses may be involved. This produces conjunctival irritation (itchy, watery eyes), redness and tearing, ear fullness and popping, itchy throat, and pressure over the cheeks and forehead. Malaise, weakness, and fatigue may also be present. The coincidence of other allergic syndromes, such as atopic eczema or asthma, and a positive family history of atopy point toward an allergic pathology. Approximately 20% of cases are accompanied by symptoms of asthma.[4]

When a clear relation between onset of pollenation and the typical rhinitis symptoms is present, the diagnosis of AR is relatively simple. However, when all of the

TABLE I. Causes of rhinitis

- Allergic rhinitis
  - Seasonal
  - Perennial
  - Perennial with seasonal exacerbations
- Nonallergic rhinitis
  - Structural/mechanical factors
    - Deviated septum/septal wall anomalies
    - Hypertrophic turbinates
    - Adenoidal hypertrophy
    - Foreign bodies
    - Nasal tumors
      - Benign
      - Malignant
    - Choanal atresia
  - Infectious
    - Acute
    - Chronic
  - Inflammatory/immunologic
    - Wegerner granulomatosis
    - Sarcoidosis
    - Midline granuloma
    - Systemic lupus erythematosus
    - Sjögren syndrome
    - Nasal polyposis
  - Physiologic
    - Ciliary dyskinesia syndrome
    - Atrophic rhinitis
    - Hormonally induced
      - Hypothyroidism
      - Pregnancy
      - Oral contraceptives
      - Menstrual cycle
      - Exercise
      - Atrophic
    - Drug-induced
      - Rhinitis medicamentosa
      - Oral contraceptives
      - Antihypertensive therapy
      - Aspirin
      - Nonsteroidal anti-inflammatory drugs
    - Reflex-induced
      - Gustatory rhinitis
      - Chemical or irritant-induced
      - Posture reflexes
      - Nasal cycle
    - Environmental factors
      - Odors
      - Temperature
      - Weather/barometric pressure
      - Occupational
  - NARES (nonallergic rhinitis with eosinophilia syndrome)
  - Perennial nonallergic rhinitis (vasomotor rhinitis)
  - Emotional factors

TABLE II. Comparison of in vivo skin tests versus in vitro serum IgE antibody immunoassay in allergic diagnosis

Skin test	Serum immunoassay
Less expensive	No patient risk
Greater sensitivity	Patient-doctor convenience
Wide allergen selection	Not suppressed by antihistamines
Results available immediately	Results are quantitative
	Preferable to skin testing in
	Dermatographism
	Widespread dermatitis
	Uncooperative children

typical rhinitis symptoms are not expressed, the diagnosis is more difficult. Chronic nasal obstruction alone may be the major symptom of PAR as a result of ongoing inflammation and late phase allergic reactions.[24]

Distinct temporal patterns of symptom production may aid diagnosis. Symptoms of rhinitis, which occur each time that the patient is exposed to a furry pet, suggest IgE-mediated sensitivity to that pet. Furthermore, patients who are sensitive to animal proteins may develop symptoms of rhinitis and asthma when entering a house, even though the animal was removed several hours earlier. Exposure to airborne allergens in the school or work environment may produce symptoms only during the week, with a symptom-free period at weekends. Likewise, vacations may be notably symptom-free.

Several processes and anomalies in presentation may complicate the diagnosis of AR. For example, the symptoms on any particular day of pollen exposure will be influenced by exposure on that day but also on previous days because of the priming phenomenon. As a consequence, at the end of the pollen season, the decline in symptoms usually takes place more slowly than the decline of pollen counts themselves.[32] In cases of perennial rhinitis, the symptoms are chronic and persistent, and patients may have secondary complaints of mouth-breathing, snoring, sinusitis, otitis media, or "a permanent cold."[33]

Nonallergic rhinitis

The most common form of nonallergic rhinitis in children is infectious rhinitis. Infectious rhinitis may be acute or chronic. Acute infectious rhinitis, such as the common cold, is usually caused by one of a large number of viruses, but secondary bacterial infection with sinus involvement may be a complication. Symptoms of chronic infectious rhinosinusitis include mucopurulent nasal discharge, facial pain and pressure, olfactory disturbance, and postnasal drainage with cough.

The symptoms of AR are frequently confused with infectious rhinitis when patients complain of a constant cold. Symptoms persisting longer than 2 weeks should prompt a search for a cause other than infection. If tests for atopy or airway disease (eg, asthma) are negative, foreign body rhinitis should be considered in the differential diagnosis. In such cases, symptoms may be acute or chronic, unilateral or bilateral, and the nasal discharge may be bloodstained or foul-smelling.

Exacerbations of rhinitis symptoms with predominant clear rhinorrhea in patients with a known history of AR may be difficult to diagnose. The difference between active infection and allergy should be made. When the history or physical examination is not diagnostic, a nasal smear should be obtained to aid in differentiation.

Allergy, mucociliary disturbance, and immune deficiency may predispose certain individuals to the development of chronic infection.[34,35] Mucociliary abnormalities may be congenital, as in primary ciliary dyskinesia,[36] Young syndrome,[37] or cystic fibrosis, or they may be secondary to infection. Similarly, immune deficiency may be congenital or acquired.

Tumors or nasal polyps as well as other conditions (eg, nasal septal deviation, adenoidal hypertrophy, hypertrophy of the nasal turbinates) can produce nasal airway obstruction. Nasal polyps are common in children with cystic fibrosis but not in children with AR. Tumors as a cause of rhinitis are very uncommon in children. Other anatomic anomalies are more common in children. Nasal septal deviation and nasal turbinate or adenoidal hypertrophy may block the flow of nasal secretions, leading to rhinorrhea or postnasal drip, as well as causing nasal blockage. The most common acquired anatomic cause of nasal obstruction in infants and children is adenoidal hypertrophy.

Children with rhinitis should also be assessed for congenital and acquired anatomic causes of nasal obstruction.[38] Reduced air flow through the nasal passages in infants may be caused by congenital choanal atresia.

Refractory clear rhinorrhea may be caused by cerebrospinal fluid leak, even in the absence of trauma or recent surgery. Any case of suspected tumor should be promptly referred to an otolaryngologist for a complete examination of the upper respiratory tract.

Diagnostic tests

Nonspecific allergy tests. Laboratory confirmation of the presence of IgE antibodies to specific allergens, such as dust mites, pollens, or animal dander, is helpful in establishing a specific allergic diagnosis, especially if the history of specific allergen exposure is not clear-cut. In many patients, it is necessary to test for specific allergens to convince the family and patient of allergic diagnosis and reinforce the importance of environmental control measures.

Although skin testing might be performed on any child of any age, children younger than l year of age may not display a positive reaction. Often the child with seasonal respiratory allergy will not have a positive test until after two seasons of exposure. Clinicians should be selective in the use of allergens for skin testing and should use only common allergens of potential clinical importance. The most useful allergens for testing in the child with perennial inhalant allergy are house dust mite (Dermatophygoides), animal danders, and fungi (molds). Allergens important in the diagnosis of seasonal AR are weed, grass, and tree pollens. Because there is significant geographic specificity with regard to pollens, the importance of these seasonal allergens varies not only by season of year but by geographic distribution. Therefore, allergens

used for skin testing must be individualized and should be selected on the basis of prevalence in the patient's geographic area of the country and the home and school environment in which they live and play.

There are two methods for specific IgE antibody testing: in vivo skin testing and in vitro serum testing[39] (see Table II for advantages and disadvantages). At the present time, properly performed skin tests are the best available method for detecting the presence of allergen-specific IgE. The skin prick, also called the puncture or epicutaneous skin test, is the preferred method of IgE antibody testing. Scratch testing has been abandoned as too traumatic. If skin prick tests are negative and allergy is highly suspected, then intradermal testing, which is more sensitive but less specific, may be used if indicated.

In vitro tests are acceptable substitutes for skin tests in the following circumstances: (1) the patient has abnormal skin, such as dermatographism or extensive dermatitis, (2) the patient cannot or did not discontinue antihistamines or other interfering medications, (3) the patient is very allergic by history and anaphylaxis is a possible risk, and (4) the patient is noncompliant for skin testing.

To avoid false-negative skin tests, short-acting antihistamine medications should be withheld for 36 to 48 hours, and long-acting antihistamines, for example, astemizole, should be withheld for 4 to 6 weeks before skin tests are performed because antihistamines suppress the skin test results.

Physicians must remember that positive tests for allergen-specific IgE themselves are not sufficient for a diagnosis of allergic disease. These tests only indicate the presence of IgE molecules with a particular immunologic specificity. A decision whether the specific IgE antibodies are responsible for clinically apparent disease must be based on the physician's assessment of the entire clinical picture. The ultimate standard for the diagnosis of allergic disease remains the combination of (1) positive history, (2) the presence of specific IgE antibodies, and (3) demonstration that the symptoms are the results of IgE-mediated inflammation.

Nonspecific allergy tests. Blood eosinophilia and total serum IgE level have been proposed as screening tests for allergies, but they have relatively low sensitivity and should not be used routinely for diagnosis of AR. The nasal secretions or sputum of patients with respiratory allergy contain increased numbers of eosinophils, which forms the basis of a useful nonspecific test (nasal smear for eosinophils), but one that will not identify any specific allergen etiology. Nasal smears for eosinophil/neutrophil counts can be useful in differential diagnosis when the diagnosis is unclear.

CONCLUSIONS

Despite the high prevalence of AR in the pediatric population, this disease is often overlooked or undertreated. Untreated AR impairs the quality of life of the child and his or her parents. Accurate and timely diagnosis of AR in children relies on awareness of the symptoms and signs of the disease and its comorbidities, including asthma, sinusitis, and otitis media. Clinicians should understand the differential diagnosis of AR in children and pursue specific diagnostic testing when indicated.

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