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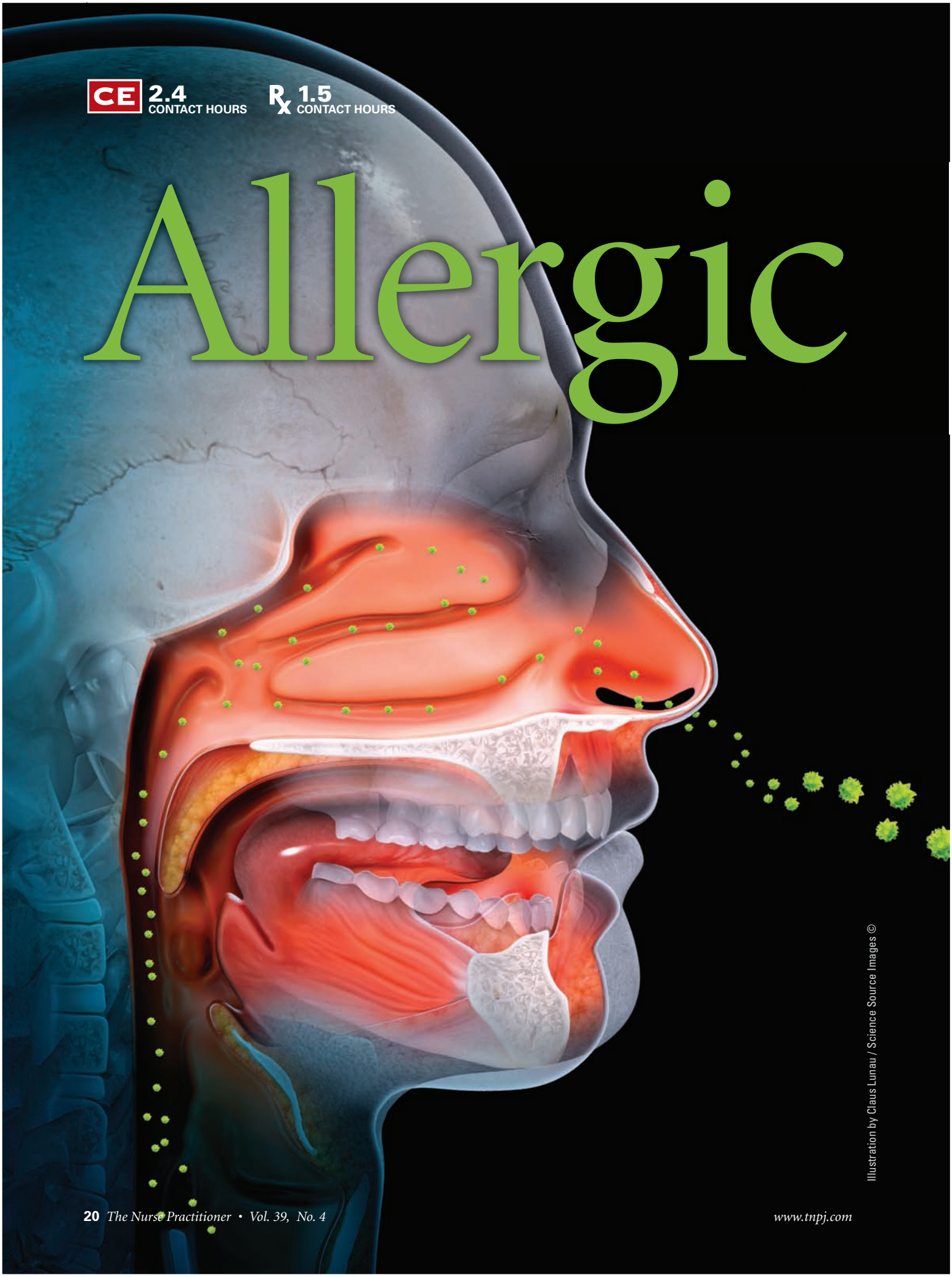
Allergic



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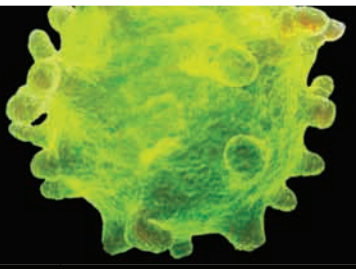
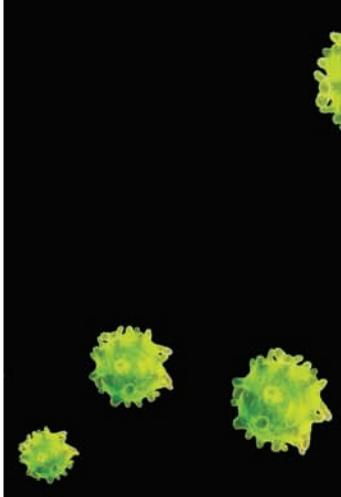


rhinitis

Diagnosis through management

Abstract: Allergic rhinitis (AR) is an immune hypersensitivity response of the nasal mucosa affecting children and adults. Patients with a genetic predisposition become sensitized to certain allergens over time with repeated exposures. This article will discuss AR from diagnosis through treatment.



By Helene J. Krouse, PhD, ANP-BC, FAAN, and John H. Krouse, MD, PhD, FACS, FAAAAI

Mr. G, a 35-year-old office worker, presents at a clinic in the late spring complaining of nasal congestion, sneezing, and watery eyes. He states that symptoms started about a month ago but are getting worse. He denies fever or enlarged, tender cervical lymph nodes. Mr. G enjoys working outdoors but noticed that symptoms worsen when he is mowing the lawn and improve in an air-conditioned setting. Mr. G was feeling so badly that he purchased an over-the-counter (OTC) cold remedy 2 days ago but reports only minimal relief of symptoms. He believes it is a respiratory infection and is requesting antibiotics for his symptoms.

Mr. G lives with his wife, two young children, and his tabby cat, Max. He does not smoke and has two to three alcoholic drinks per week. He is doing some renovations in his basement that has a moisture problem. Mr. G participates in moderate exercise three times a week and tries to eat healthy, since he was recently diagnosed with mild hypertension and is taking a once-daily diuretic. His family history is unremarkable for respiratory, cardiovascular, or immunologic disorders. In the last 6 months, one of his sons was diagnosed with atopic asthma.

Keywords: allergic rhinitis, allergy, hypersensitivity disorder, management

Vital signs: Temperature 99° F (37.2° C), pulse 76, respirations 16, BP 130/78.

Physical exam: Head–Normocephalic

Eyes–Conjunctivae pale, excessive tearing, and mild periorbital edema noted bilaterally

Ears–Tympanic membranes mobile

Nose–Pale, boggy mucous membranes, clear rhinorrhea bilaterally, inferior turbinates moderately swollen, no polyps

Oropharynx–Clear, no erythema

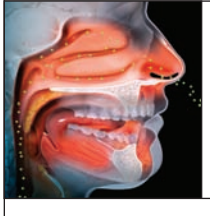
Neck–Supple, no tenderness or adenopathy

Lungs–Clear to auscultation bilaterally

Epidemiology and pathophysiology

In the United States, approximately 8% of adults and 11% of children suffer from respiratory allergies annually.[1] It appears that allergic rhinitis (AR) has been increasing in prevalence globally, now affecting between 10% and 30% of the population worldwide.[1,2] It is often associated with comorbid conditions, including asthma, rhinosinusitis, eczema, and gastric reflux.

AR is a hypersensitivity disorder of the nasal mucosa that represents an abnormal immunologic response characterized by increased nasal inflammation.[3] This response is mediated by immunoglobulin E (IgE). It is an immune condition that occurs in a patient with a genetic predisposition to develop such a response and who has been sensitized through repeated exposure to certain antigens. This genetic predisposition is known as atopy, and the person who is able to mount this immune response is referred to as atopic.



In the United States, approximately 8% of adults and 11% of children suffer from respiratory allergies annually.

In an individual previously sensitized to an allergic antigen (or allergen), repeated exposure to that antigen will result in a characteristic immune response in which these allergens bind to specific antibodies on the surface of inflammatory cells (known as mast cells) and trigger the release of various chemical mediators, the most important of which is histamine. Histamine then binds to receptors on the surfaces of target cells, generating the well-recognized symptoms of AR: sneezing, itching, nasal discharge (rhinorrhea), and nasal congestion. This process occurs rapidly, with many patients noting the onset of symptoms within 5 minutes of allergen exposure.

In addition to the rapid and immediate release of histamine, stimulated mast cells also release other important agents, including leukotrienes, which are potent mediators of inflammation that can prolong symptoms. One effect of leukotrienes is to increase the presence of inflammatory cells known as eosinophils, which are involved in sustaining the clinical symptoms seen in AR. The disease of AR is therefore biphasic, with an early phase response primarily triggered by histamine and a later phase response that is sustained by leukotrienes and activated eosinophils.[4] Treatment strategies for AR rely on an appreciation of these pathophysiologic mechanisms.

As described in the United States, AR is commonly divided into two categories: seasonal allergic rhinitis (SAR), which is related to the seasonal release of pollens from plants and lasts for the discrete periods in which those pollens are present, and perennial allergic rhinitis (PAR), which occurs throughout the year and is often attributed to perennial allergens, such as dust mites, animal dander, and molds. In temperate climates, SAR can generally be seen in three discrete seasons timed with the release of certain pollens. In broad terms, tree pollens are generally seen in the early spring months, grass pollens in the summer, and weed pollens from the late summer through the early fall. In more southern climates, prolonged warm temperatures often blur these seasonal distinctions and lead to prolonged symptoms that can mimic PAR. It is important to note that patients with SAR are usually more symptomatic outdoors while pollens are present, and patients with PAR are often more symptomatic indoors when exposed to allergens that are present year-round in the indoor environment.[5]

Internationally, AR is classified using the Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines based on its temporal relationship as either intermittent AR, which occurs sporadically throughout each week or for only a short period of time annually, or persistent AR, which involves symptoms that are present throughout most of the year and on a daily basis.[3] In addition, the ARIA guidelines characterize the severity of AR based on its impact on quality of life and daily function. The U.S. has traditionally preferred classifying AR into seasonal versus perennial disease, so that terminology will be used in this article.

Assessment and clinical presentation

Eliciting a thorough history is vital to the nurse practitioner's (NP's) understanding of underlying pathology and contributing environmental conditions. Since AR has a strong genetic component, family history and age of symptom onset provide information critical to making an accurate diagnosis. Nasal symptoms are often accompanied by other irritating symptoms, such as tearing, watery eyes, palatal itching, or aural fullness.[4] Specific questions regarding the onset of

symptoms must be included to identify specific triggers (exposure to pollens or animal dander) and environmental conditions (seasonal variability, geographic areas, or outdoor activities). (See *Patient history questions to consider.*)[5] Seasonal variability in patient symptoms helps the NP confirm an AR diagnosis. Respiratory irritants such as cigarette smoke and air pollutants can further exacerbate AR and should be included in the patient's health history.

On physical exam, the NP should observe the face and eyes for any periorbital edema, puffiness, infraorbital darkening of the skin (known as "allergic shiners"), and fine creases in eyelids (known as "Dennie-Morgan lines"). The most common nasal findings on examination include enlarged inferior turbinates, pale mucosa with gray-to-blue appearance, and clear, watery discharge. The NP may also note a transverse nasal crease at the tip of the nose, known as the "nasal salute" or "allergic salute," which is an indicator of AR resulting from excessively rubbing the nose. The ear canal and tympanic membrane should be examined, as otitis media is a frequent comorbid condition. The tympanic membrane is assessed for mobility, inflammation, and retraction as well as any drainage. Examination of the oral cavity may reveal enlarged tonsils in children as well as a cobblestone appearance of the posterior oropharynx.

Several other conditions present with symptoms and physical findings similar to AR, and providers should consider these as potential differential diagnoses. Common differential diagnoses include nonallergic rhinitis and infectious rhinitis (upper respiratory infections and acute rhinosinusitis).[4,5] The presentation of nonallergic rhinitis may be very similar to AR with either variable or persistent symptoms. The main distinction between AR and nonallergic rhinitis is the absence of an IgE-mediated immune response in the latter, which can be determined through various diagnostic methods (discussed below). The NP should also consider whether an infectious process is present and should evaluate the patient for viral and bacterial upper respiratory infections, including acute rhinosinusitis. The most common symptoms of acute infectious rhinosinusitis are nasal congestion, pressure and pain, headache, and purulent nasal discharge.[5] Most upper respiratory infections are caused by viruses and usually resolve spontaneously within 7 to 10 days. In patients with acute bacterial rhinosinusitis, symptoms and physical findings may persist beyond 10 days and even become worse after an initial improvement in symptoms, requiring the NP to further evaluate the condition and the treatment prescribed.[6]

Diagnostic testing

When the clinician suspects a diagnosis of AR, specific testing methods can be used to confirm or refute the diagnosis.

Patient history questions to consider

1. At what age did your symptoms begin?
2. Do any members of your family have allergies?
3. Do your symptoms improve or resolve in different geographic locations (for example, travel to other parts of the country)?
4. How often do symptoms occur? How long do these symptoms last?
5. Do your symptoms occur at the same time each year?
6. Do your symptoms occur in specific places (for example, home, office, school)?
7. Do your symptoms occur in the presence of specific triggers (for example, cats, trees, dust)?
8. Do your symptoms occur when you engage in specific activities (for example, gardening, mowing the lawn, or working in the basement)?
9. What do you think is causing your symptoms?
10. Do you have other respiratory conditions, such as asthma, bronchitis, or emphysema?

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.

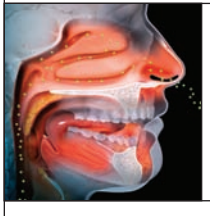
When identification of specific sensitivities is desired, there are several procedures available with varying degrees of sensitivity and specificity that can be completed either with skin testing methods or with blood tests.

Total serum IgE: One simple screening test that is often utilized as a gross measure of the presence of AR is the assessment of total IgE level in the serum. When highly elevated, this blood test suggests the presence of allergy (for example, total IgE greater than 400 units/mL) and is correlated with the presence of symptomatic allergic disease. Unfortunately, in patients with lower levels of total IgE, the correlation is less robust. Even in patients with extremely low levels of total IgE (less than 10 units/mL), up to one third will have significant allergy when assessed through more sensitive methods.[7] In other words, even though high levels of total IgE are predictive of significant allergy, low levels do not rule out the presence of clinically important AR. Patient screens with total IgE are therefore only helpful in confirming the presence of allergy when levels are very high.

Skin testing: The most commonly performed and most sensitive diagnostic methodology used for assessing AR is skin testing. In skin testing, small amounts of allergens that the patient is suspected of being allergic to can be introduced into the skin through two methods: superficially through prick testing and intradermally through the creation of a small wheal of diluted antigen. In sensitized patients, this antigen will bind to the surface of mast cells, causing the release of histamine into the local tissues and the induction

of swelling and redness at the testing site (wheal and flare response). The extent of this response can be graded, and significant local reaction confirms specific allergic sensitivity. Skin testing is safe, sensitive, specific, and rapidly and easily performed in the office setting. The patient is informed of the skin test results and what environmental allergens to avoid. The results are also used to prepare sera for specific immunotherapy if beneficial for the patient.[8]

In vitro testing: Another method for assessing individual allergen sensitivities is through the measurement of specific IgE levels to suspected antigens from the patient's serum. In this methodology, often referred to generically as radioallergosorbent (RAST) testing, the patient's serum is incubated with tagged antibodies of suspected allergens that can be measured to precisely assess the amount of those specific antigens in the serum.[9] The presence of these specific IgE molecules and their absolute level in the serum are precise indicators of both the patient's sensitivity to that antigen and the degree of sensitization. As with skin testing, these quantitative results can be used both to counsel the patient on avoidance and to prepare a serum vial for immunotherapy. While in vitro testing will generally offer excellent results, skin testing is a more sensitive and preferred method for assessing AR.[10]



The mainstay of AR treatment is the use of medications to control and alleviate symptoms.

Other methods: While the above three methodologies are the most commonly used in diagnostic testing for suspected AR, other methods can sometimes be employed. Nasal swabs can be taken to examine for the presence of eosinophils, which are generally elevated in the nasal mucosa of patients with AR. This test is both poorly sensitive and poorly specific. In addition, the nasal mucosa can be challenged directly with suspected antigens, and the patient's response can be assessed both through induction of symptoms and physiologic measures.[11] This procedure is time-consuming, often inaccurate, and is not practical in the general clinical setting. Elevation of eosinophils in the patient's serum on a complete blood cell count is also associated with allergy, although again, this is a poorly specific measure for diagnosing AR.

Treatment

A comprehensive approach for treating AR includes of environmental control measures, pharmacotherapy, education, and potentially immunotherapy to maximize outcomes for the patient. A complete discussion of therapeutic strategies for AR can be found in the 2008 practice parameters of the Joint Council of Allergy, Asthma, and Immunology.[5]

Environmental control measures

When practical, patients should attempt to decrease their exposure to known allergic triggers through avoidance whenever possible. Although environmental controls can be useful, patients often find them difficult to implement. Allergens most problematic in the home include animal dander, dust mites, and molds. For example, cat allergy is one of the most potent sensitivities; however, families are often reluctant to remove pets from the home.[12] Simply keeping a pet out of the bedroom can reduce antigen exposure in that area and decrease symptoms. Another strategy is to bathe pets regularly to reduce dander and eliminate allergens that they may bring in from the outdoors. In individuals allergic to dust mite antigens, an allergen-resistant mattress and pillow covers along with high-efficiency air filtration (HEPA filters) can help reduce exposure. Minimizing dust-collecting objects such as stuffed toys, throw pillows, and carpeting can also reduce allergens in the home.[13]

Indoor humidity and dampness can foster mold growth, particularly in basements, kitchens, and bathrooms. Patients can reduce mold levels by sealing openings to prevent mold spores from entering the home and using dehumidifiers, ceiling exhaust fans, and air conditioning to lower humidity. Additional measures that can help reduce indoor levels of mold and pollen include removing clothing when entering the home and showering after engaging in outdoor activities. Other environmental measures that might be helpful are removing carpeting and replacing with hardwood floors, frequently changing filters on heating and cooling units, keeping living areas clutter-free, and properly sealing windows and doors.

Pharmacotherapy

The mainstay of AR treatment is the use of medications to control and alleviate symptoms. Healthcare providers recommend and prescribe a range of topical and systemic medications to patients with allergic symptoms, and these various classes of pharmacotherapeutic agents demonstrate differing degrees of symptom relief based upon their route of administration and their mechanism of action (see *Classes of medications used to treat AR and their relative efficacy*).[14]

Antihistamines: Oral antihistamines were first introduced in the 1940s and became widely used in the 1950s. These medications work by attaching to histamine receptors on the surface of target cells in the nose and other tissues

Classes of medications used to treat AR and their relative efficacy

Agent	Sneezing	Itching	Congestion	Rhinorrhea	Eye Symptoms
Oral antihistamines	++	++	-	++	++
Nasal antihistamines	+	+	++	+	-
Intranasal corticosteroids	++	++	+++	++	+
Leukotriene modifiers	+	+	+	+	+
Oral decongestants	-	-	++	-	-
Nasal decongestants	-	-	+++	-	-
Nasal mast-cell stabilizers	+	+	+	+	-
Topical anticholinergics	-	-	-	+++	-

+++ = marked benefit; ++ = substantial benefit; + = some benefit; +/- = minimal benefit; - = no benefit

Adapted from: Krouse, JH, Derebery MJ, Chadwick SJ (eds). Managing the Allergic Patient. New York: Saunders; 2008.

and deactivating these receptors. Antihistamines have well-known adverse reaction profiles, which have changed significantly over the years. Antihistamines can be broadly classified into two categories: earlier or first-generation antihistamines, which were in general use until the late 1990s, and newer or second-generation antihistamines, which have largely supplanted earlier drugs in the treatment of AR.[15]

While first-generation antihistamines demonstrated reasonable efficacy in treating allergic symptoms in both children and adults, they were often poorly tolerated due to their significant adverse reactions. Two major groups of adverse reactions accompanied the use of these agents: central nervous system (CNS) effects, such as sedation, cognitive impairment, and psychomotor dysfunction and anticholinergic effects, such as blurred vision, dry mouth, urinary retention, and increased mucus tenacity. Drowsiness and related symptoms can be disabling, even in the absence of perceived sedation, resulting in poor work, school, or psychomotor performance. Examples of first-generation antihistamines include diphenhydramine, chlorpheniramine, triprolidine, and promethazine.

In order to retain clinical effectiveness, yet at the same time decrease the frequency of unwanted adverse reactions, newer antihistamines have been developed that are currently the preferred agents for treating AR. These second-generation antihistamines possess excellent efficacy, yet have a much more advantageous safety profile, accompanied by little or no sedation or adverse anticholinergic reactions. Since some patients may still demonstrate sensitivity to the sedating effects of second-generation antihistamines, it is important to monitor patients for these signs and symptoms. Examples of currently available second-generation antihistamines include loratadine, desloratadine, fexofenadine, cetirizine, and levocetirizine.

In general, while antihistamines are effective in treating the symptoms of sneezing and itching among patients with AR, they demonstrate lesser efficacy in treating rhinorrhea and have little or no effect on nasal congestion. Antihistamines are often combined, therefore, with oral decongestants in patients with nasal congestion. In addition, other classes of agents, notably intranasal corticosteroid sprays, possess robust efficacy in the treatment of nasal congestion without the adverse reaction profile that often accompanies the use of oral decongestants.

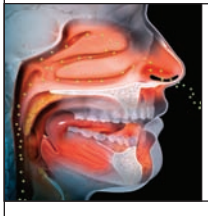
Finally, over the past decade, topical nasal antihistamines have been available for treating AR and demonstrate both excellent efficacy and rapid onset of action. In addition, topical antihistamines are effective in reducing nasal congestion as well as in relieving symptoms of sneezing and itching. In comparative studies, topical antihistamines have been demonstrated to be more effective than their oral counterparts.[16] Two agents are available for prescription in the United States: azelastine and olopatadine. Both can be associated with an unpleasant taste in some patients, and somnolence in sensitive individuals.

Decongestants: Decongestant medications have been used since the middle of the 20th century and are recommended specifically to reduce nasal congestion in patients with AR. These medications stimulate alpha-adrenergic receptors in the nose as well as throughout the body and decrease blood flow and engorgement of the vascular tissues that create nasal obstruction. They are available in both topical and oral forms.

Oral decongestants can be used alone to treat patients who complain of isolated nasal obstruction but are often combined with oral antihistamines to treat other common symptoms of AR, such as sneezing and itching. Two oral decongestants are currently available in the United States:

pseudoephedrine and phenylephrine. Since pseudoephedrine can be chemically converted into methamphetamine, drugs containing pseudoephedrine are now held behind the pharmacy counter, and identification/signatures are commonly necessary for patients to receive this drug.[17] Phenylephrine is available without this restriction and is freely available OTC; however, it is somewhat less effective than pseudoephedrine.

Oral decongestants are often accompanied with unpleasant adverse reactions that can limit their use. Due to their general alpha-adrenergic effects, both CNS and cardiovascular symptoms are experienced by many patients. CNS effects are due to the stimulant properties of these medications and include anxiety, nervousness, tremulousness, irritability, and insomnia. Cardiovascular symptoms include tachycardia, palpitations, hypertension, and irregular heartbeat. Other systemic effects include nausea, vomiting, increased intraocular pressure, and urinary retention. Patients at risk for CNS, cardiac, or ocular diseases should avoid the use of oral decongestants.[4] It is wise to limit the use of these medications to healthy individuals for the shortest time possible at the lowest effective dose.



Newer topical corticosteroids have minimal systemic absorption and few adverse reactions.

Decongestants can also be used topically in order to provide a rapid decrease in nasal obstruction. When sprayed into the nose, topical decongestants will reduce blood flow to nasal vascular tissue, resulting in a decrease in congestion and an improvement in airflow. Two medications are in common use in the United States: oxymetazoline and phenylephrine. While topical decongestants provide rapid improvement in nasal blockage, they are frequently accompanied by tachyphylaxis and dependency, even after short-term use. This dependency is significant and can create severe nasal dysfunction. For this reason, topical decongestants should never be used for more than 3 to 5 days consecutively.[15]

Mast cell stabilizers: A mast cell stabilizer, cromolyn sodium, is available for topical use in the treatment of patients with AR. This medication inhibits the release of histamine and other mediators from mast cells and results in decreased histamine stimulation of target cells. In order for mast cell stabilizing medications to be effective, they must be given prior to exposure. Unfortunately, while cromolyn sodium is a safe medication, it is of limited efficacy and only has a mild effect on reducing allergic symptoms.[15] In addition, it has a very short half-life and must be given four times daily in order to have appropriate pharmacologic effect. Mast cell stabilizers, therefore, have limited practical use in treating the symptoms of AR, especially since more effective medications are widely available.

Corticosteroids: Corticosteroids are potent anti-inflammatories that are highly effective in reducing the symptoms of AR. They are beneficial in treating all symptoms associated with AR and have robust effects on nasal congestion. While corticosteroids may be given both systemically and topically to treat AR, parenteral administration is not commonly utilized in routine practice due to the higher incidence of significant adverse reactions and risks. In rare cases of severe disease, systemic use of corticosteroids may be warranted.

Supported by current guidelines, topical corticosteroids are often employed as the primary, first-line treatment method for many patients with AR.[18] Newer topical corticosteroids have minimal systemic absorption and few adverse reactions. They work as anti-inflammatory agents, resulting in the reduction of many inflammatory mediators involved in the allergic response. Examples of commonly used topical corticosteroids include beclomethasone dipropionate, triamcinolone acetate, budesonide, fluticasone propionate, mometasone furoate, fluticasone furoate, and ciclesonide. Newer agents such as mometasone furoate and fluticasone furoate have been shown to be safe and are approved for use in children 2 years of age and older.[19,20]

Topical corticosteroids are generally well tolerated by patients, although they can be associated with nasal dryness, stinging, or epistaxis. Proper instruction in the use of nasal corticosteroids, with application directed to the lateral portion of the nasal mucosa, often results in fewer adverse reactions and decreased bleeding. In addition, while patients may sometimes notice marginal improvement in symptoms within the first day of use, they will generally require 7 to 10 days to notice a robust clinical effect.

Leukotriene modifiers: Leukotriene modifiers are used in treating AR and other airway inflammatory diseases, such as asthma. They decrease late-phase allergic symptoms through interfering with the attachment of leukotrienes to receptors on target cells. The only drug of this class approved for use in AR in the United States is montelukast. Montelukast is effective in reducing all symptoms of AR and has potency roughly equal to that of an oral antihistamine. It is safe, well tolerated, and approved for use in children age 2 years and older for SAR and in children 6 months of age and older for PAR.[21]

Topical anticholinergics: A portion of the rhinorrhea experienced by patients with AR is due to parasympathetic stimulation of the nasal mucosa. In these patients, topical anticholinergic medications may reduce the volume of clear rhinorrhea but have no measurable effect on sneezing, itching, or nasal congestion. The only topical anticholinergic spray available for nasal use in the United States is ipratropium bromide.

Nasal irrigations: Although not a pharmacotherapeutic method, nasal irrigation is an effective topical treatment for nasal symptoms, as confirmed by a recent systemic review and meta-analysis.[22] These irrigations consist of rinsing the nose with saline or salt water in order to mechanically remove irritants and allergens from the nasal passages. In order to reduce the risk of infection from environmental pathogens in tap water, patients should only make nasal irrigation solutions from sterile, distilled, or previously boiled water.[23] An ameba, *Naegleria fowleri*, has been found in freshwater lakes and rivers in warmer climates. If water containing this ameba is used for nasal irrigations, it can migrate to the brain area and is usually fatal; therefore, patients should be instructed to never use untreated tap water from the sink for nasal irrigations.[23] Devices used to perform nasal irrigations include the bulb syringe, neti pot, and spray bottles. Patients should also be advised to rinse their irrigation devices with sterile, distilled, or boiled water after each use in order to reduce contamination.[23]

Patient education

AR can be effectively managed if patients understand causes and triggers of their symptoms and utilize available treatment strategies. An effective method to prevent symptoms of AR is avoidance of allergic stimuli. This option may not be practical, however, especially when symptoms result from naturally occurring exposures, such as plant pollination or animal dander. When patients understand what is causing or triggering their symptom cascade, they can learn strategies to avoid, eliminate, or minimize exposure to the allergen and better control their environment. Through education, patients can feel empowered in partnering to help effectively manage AR. These strategies include approaches to help manage both indoor and outdoor exposures.

Immunotherapy

For patients whose medical management and environmental control methods have been less effective than anticipated, immunotherapy is the next treatment strategy that can be useful in patients with AR. Immunotherapy involves the sequential administration of increasing doses of antigens to which patients are found to be allergic on testing, with the goal of inducing hyposensitization and decreased response to those antigens on exposure. Immunotherapy is generally administered through subcutaneous injections in the provider's office, although there is increasing interest in the United States regarding delivery of immunotherapy through sublingual administration. Sublingual immunotherapy is currently considered investigational in the United States, although several commercial products are currently under review by the FDA.[24]

Both sublingual and subcutaneous immunotherapies are generally effective, safe, well tolerated by patients, and can be useful even as an adjunct to pharmacotherapy and avoidance. Immunotherapy has been extensively studied, and there is robust evidence supporting its efficacy and safety.[25,26]

Case study follow-up and plan

Based on Mr. G's symptoms and history, he was diagnosed with AR. Acute rhinosinusitits, upper respiratory infection, and nonallergic rhinitis were differential diagnoses that were also considered for Mr. G. He was managed by the NP using a 3-pronged treatment approach that included patient education, environmental controls, and pharmacotherapy. The NP initially discontinued Mr. G's use of the OTC medication, since it contained an oral decongestant that raises BP. He was instructed on symptom recognition, differences between AR versus respiratory infections, and the negative consequences of overprescribing antibiotics. He was shown how to properly perform nasal irrigations and practiced the technique in the office. He was also educated on environmental measures to help reduce, control, and eliminate exposure to specific allergens. The NP prescribed a nonsedating, once-daily antihistamine along with an intranasal corticosteroid to reduce his bothersome nasal symptoms. Mr. G will follow up in 2 weeks to evaluate treatment effectiveness. Depending on his responsiveness to pharmacotherapy and supportive measures, he may be further evaluated by allergy testing and initiated on immunotherapy. By employing a multifaceted approach, the NP can be most effective in helping Mr. G manage his symptoms and improve his overall quality of life. NP

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