

Allergic Rhinitis

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KEYWORDS

- Allergic rhinitis • Epidemiology • Diagnosis
- Immunotherapy • Environmental control

Key Points

- Physicians evaluating patients for functional nasal problems should be familiar with the diagnosis and treatment of allergic rhinitis.
- Allergic rhinitis is a common disorder that causes symptoms of rhinorrhea, sneezing, nasal itching, and nasal congestion.
- Allergic rhinitis also affects quality of life, sleep, school performance, and productivity.
- Management options for allergic rhinitis include pharmacotherapy, environmental control, and immunotherapy.
- Turbinate reduction may be useful in selected patients with allergic rhinitis.
- Nasal congestion secondary to allergic reaction has the potential to negatively affect the functional outcome of nasal esthetic procedures that narrow the nasal valve.

Facial plastic surgeons need to be familiar with common inflammatory conditions of the nose, as these affect nasal function. Frequently, nasal obstruction has both structural and inflammatory contributions and recognition of both is important. Counseling patients about coexisting nasal inflammation helps establish more realistic expectations of nasal surgical results and can be used to guide patients toward other effective treatments. One of the most common sources of nasal inflammation is allergic rhinitis. Although not life threatening, allergic rhinitis has a substantial affect on a patient's quality of life, sleep, school performance, and productivity.¹ Physicians should also be aware of the association of allergic rhinitis with other conditions, such as asthma. Asthma is both underdiagnosed and suboptimally controlled in the United States.² This article focuses on the epidemiology, diagnosis, and treatment of allergic rhinitis. As there are articles elsewhere in this issue on the related topics of nonallergic rhinitis, allergic

pharmacotherapy, and the united airway, these topics will not be covered in detail.

DEFINITION AND GENETICS OF ALLERGIC RHINITIS

Allergic rhinitis is clinically defined in the 2008 Allergic Rhinitis and its Impact on Asthma (ARIA) Guidelines as “a symptomatic disorder of the nose induced after allergen exposure by an IgE-mediated inflammation.”³ Allergic patients have a genetic tendency to produce an inflammatory response to particles that are normally harmless. It is the inflammation that causes their symptoms. As the inflammation is unnecessary for nonpathogenic particles (such as pollen), these are described as hypersensitivity reactions. Atopy is the genetic predisposition to develop allergic hypersensitivity reactions. Atopic disease generally causes local inflammation at the surface of exposure and is classified as such. Examples

Financial disclosures: None.

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Facial Plast Surg Clin N Am 20 (2012) 11–20

doi:[10.1016/j.fsc.2011.10.002](https://doi.org/10.1016/j.fsc.2011.10.002)

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are allergic conjunctivitis, allergic rhinitis, allergic asthma, atopic dermatitis, and food allergies. Atopic individuals with one allergic condition tend to be at risk for others.

The underlying genetics of allergic individuals is not well understood but appears to be complex. Initially, it was speculated that the genetics of allergy were based in alterations of the inflammatory process. Studies that looked for particular changes in the genetic code (single nucleotide polymorphisms or SNIPs) targeted the components of allergic inflammation. Allergic individuals were found to have more SNIPs in genes coding for many components of allergic inflammation, including interleukin (IL)-4, IL-13, and T-cell receptors, than nonallergic controls.⁴ More than 100 SNIPs have been identified as more prevalent in allergic populations than in controls; but, individual SNIP studies often have poor reproducibility.⁴ Larger studies that were hypothesis independent (such as Genome Wide Association [GWA] studies) compared thousands of known human SNIPs in allergic versus nonallergic populations. GWA studies have been mostly performed in allergic asthma. These studies identified different gene candidates that tended not to be linked directly to the allergic inflammatory cascade.⁵ It is likely that a combination of factors of innate immunity and variable changes in the regulation of inflammation may both contribute toward allergic hypersensitivity. Genetically, allergic rhinitis is a heterogenic disease.⁵

This is perhaps best illustrated in atopic dermatitis. A defect in the protein fillagrin has been identified in about one-third of individuals with atopic dermatitis.⁶ Fillagrin is a protein that helps connect or seal the outer layer of keratinocytes, and it is speculated that the genetic “loss of function” make the skin barrier more porous. One could speculate that allergic disease may require a combination of several different factors in a single individual, explaining the heterogeneity of disease. An individual with faulty innate immunity (a barrier defect like fillagrin) combined with a genetic tendency promoting inflammation may manifest allergic disease. Additionally, allergic individuals must also recognize allergen epitopes with preformed HLA, T-cell, and B-cell receptors, which may contribute to why different allergies develop.

The importance of the variety of genetic findings is that patients with similar allergic phenotypes (such as allergic rhinitis to grass pollen) may have different underlying genetics. In allergy, studies tend to differ by ethnicity and region and commonly contradict each other. Also, allergic and nonallergic rhinitis likely occur across a continuum rather than being distinctly separate processes.

BASIC IMMUNOLOGY OF ALLERGY

Although there are 6 types of Gel and Coombs hypersensitivity reactions, the Gel and Coombs Type I reaction (immunoglobulin E [IgE]-mediated hypersensitivity) is classically seen in allergic disease. During sensitization, an allergen is recognized by an antigen-presenting cell (APC) and presented to a T-helper cell lymphocyte. The allergic patient is required to have specific HLA receptors on the APC and a specific T-cell receptor for this communication to take place. T-helper cells that are biased toward promoting allergic inflammation are called TH2 cells.⁷ TH2 cells present the allergen (or allergic epitope) to a B lymphocyte, which must also have a B-cell receptor for that specific allergen. In the presence of allergic cytokines (such as IL-4) the B cell can change into an IgE-producing plasma cell for that particular allergen. The IgE produced travels through the circulation and tissue to bind to IgE receptors on mast cells and basophils.

If a patient is reexposed to the allergen and has allergen-specific IgE (sIgE) antibodies bound to their mast cells, it may cause the mast cell or basophil to degranulate, releasing inflammatory mediators, such as histamine. Histamine binds to histamine receptors on endothelial cells and vascular smooth muscle causing vasodilation and increased permeability.⁸ The patient experiences rhinorrhea and nasal congestion. Other mediators promote more inflammation such as IL-5, which promotes eosinophilia, and leukotrienes, which recruit more inflammatory cells. The promotion of more inflammation is balanced by factors that down-regulate inflammations, such as IL-10. Down-regulation appears to be partially controlled by specialized T lymphocytes (T-regs).⁹

EPIDEMIOLOGY OF ALLERGIC RHINITIS

Allergic rhinitis is a common condition in the United States and the world and is increasing in developed countries.¹⁰ Four relevant concepts to know about the epidemiology of allergic rhinitis are:

1. Prevalence
2. Cost
3. The “allergic march”
4. The “hygiene hypothesis”.

In the United States, the 2009 National Health Interview Survey conducted by the Centers for Disease Control and Prevention reported that 17.7 million adults or 7.8% of those surveyed had been diagnosed with “hay fever” in the preceding 12 months¹¹; 7.2 million children were reported to have “hay fever” and 8.2 million

(11%) were reported to have “respiratory allergy” in the preceding 12 months.¹² There is a difference between a survey for respiratory allergies and having a history and positive allergy test, however. In Sweden, by questionnaire alone, 14.2% were allergic as compared with 9.1% who were positive by questionnaire and skin test.³

The direct cost of allergic rhinitis includes medication and physician visits. In 2011, Meltzer and Bukstein¹³ estimated the direct medical cost of allergic rhinitis at \$3.4 billion in the United States. Indirect costs of missed work and lost productivity are more difficult to quantify. Given the quality of life impairment, the indirect costs are also likely to be substantial.

Because allergic rhinitis occurs as a result of genetic predisposition and environmental exposure, there is an accumulation of evidence to support the allergic or atopic “march.” This is the concept that atopic individuals often have eczema and food allergies in infancy followed by allergic rhinitis and asthma, which peaks in teenagers and young adults.¹⁴ The specifics of the allergic march seem to vary substantially in different cohorts, however.

A final important epidemiologic concept is the “hygiene hypothesis.”¹⁵ This is the controversial theory that infections early in life are protective against the later development of atopic disease. The theory is that T cells are more biased toward allergic inflammation at birth and early infections change their bias toward a nonallergic inflammatory pattern (TH1 cytokine profile) more appropriate for viral and bacterial infections. There are large well-done studies that both support and refute this theory.^{15,16}

DIAGNOSIS OF ALLERGIC RHINITIS

History

A patient’s symptom history is the most important element in determining allergic rhinitis. Allergy tests are frequently positive in individuals without significant clinical manifestations, and allergy tests do not make the diagnosis in isolation. Unfortunately, many of the symptoms in allergic rhinitis are also present in nonallergic rhinitis and chronic rhinosinusitis. The primary symptoms of allergic rhinitis are sneezing, nasal obstruction, rhinorrhea, and nasal itching.³ Anterior rhinorrhea may be a more specific sign of allergic rhinitis than post-nasal drip alone.¹⁷

Allergy should be suspected if symptoms are provoked by exposure to known allergens. If symptoms are exacerbated during a certain time of the year, such as when pollen counts are elevated, this would suggest allergy, although the

positive predictive values of specific patient-reported exposures are disappointing.¹⁸ Patients allergic to dander may also provide a history of symptoms when exposed in a home or building where the animal resides. Other allergic patients have symptoms most of the year. Most patients with allergic rhinitis are polysensitized, which complicates pairing their history with patterns of exposure.³

Allergic rhinitis is frequently divided by whether the symptoms are present intermittently or persistently. Classically, allergic rhinitis is divided into seasonal allergic rhinitis (SAR) or perennial allergic rhinitis (PAR); however, the ARIA guidelines recommend intermittent allergic rhinitis (IAR) and persistent allergic rhinitis, as they concluded that there were too many exceptions to the seasonal model, especially with dust mites and molds.³ Persistent allergic rhinitis is defined as more than 4 consecutive days per week or more than 4 consecutive weeks.¹⁹ IAR tends to have symptoms of sneezing, itching, and rhinorrhea, whereas PAR’s hallmark is nasal obstruction.

Severity of symptoms varies in allergic rhinitis and is subjective. The ARIA guidelines classify allergic rhinitis as moderate or severe when sleep disturbance or impairment of daily activities, work, or school is present (**Table 1**).³

Coexisting symptoms of wheezing or conjunctival irritation may help differentiate allergic rhinitis from nonallergic disease, as well as affect evaluation and treatment decisions. The patient’s history of other atopic disorders, such as childhood food

Table 1
ARIA classification of allergic rhinitis

Intermittent	Symptoms are present <4 days a week or <4 consecutive weeks
Persistent	Symptoms are present >4 days per week and more than 4 consecutive weeks
Mild	None of the “Moderate/Severe” criteria present
Moderate/Severe	One or more of the following are present: Sleep disturbance Impairment of daily activities, leisure, and/or sport Impairment of school or work Troublesome symptoms

Adapted from Bousquet J, Khaltaev N, Cruz AA, et al. Allergic Rhinitis and its Impact on Asthma (ARIA) 2008 update (in collaboration with the World Health Organization, GA(2)LEN and AllerGen). Allergy 2008;63(Suppl 86):8–160.

allergy or atopic dermatitis, may also support the diagnosis of allergic rhinitis.

A family history of allergies may also be useful, but allergic rhinitis does not follow simple Mendelian inheritance. Twin studies have found the proband concordance of asthma in monozygotic twins to be only 0.28 to 0.63.²⁰ In a large cohort in Sweden, family history of allergy had an odds ratio of 1.3 in predicting a positive skin-prick test in the child.¹⁰

Physical Findings in Allergic Rhinitis

Anterior rhinoscopy may be useful in considering allergic rhinitis, but physical findings are nonspecific and may be present intermittently.²¹ Inferior turbinates are generally enlarged and described as pale or boggy, but the specificity of these observations has not been carefully examined. Mucus may be more abundant or seen stranding between the turbinate and septum. Polyps are also a sign of chronic nasal inflammation, but curiously are not seen more frequently in allergic patients.²²

During acute attacks, allergy sufferers have been observed to wrinkle their face and wipe their nose, especially children. A supranasal tip crease may develop from frequently lifting the nasal tip.²³ Fine wrinkles on the lower eyelid are frequently present. Other visual findings include venous congestion and swelling of the lower lids, which give the patient the appearance of being chronically tired. The conjunctiva may have prominent vessels or thickening of the scleral surface of the eyelids. Increased mucus secretion from the eye may be present.²³ Some have observed longer eyelashes in allergic patients, which may help protect the eye from allergens.

Oral pharyngeal findings may include mucus, which tends to drain on the sides. Erythema and edema of the posterior pharyngeal wall may be observed and this is also sometimes greatest on the lateral edges.

Inhaled allergen challenges have produced laryngeal edema and stranding mucus.²⁴ Auscultation for wheezing is also recommended if the patient is suspected of being allergic.

Testing for Allergic Rhinitis

The diagnosis of allergy is primarily a clinical diagnosis, as positive allergy tests are frequently seen in individuals without allergic clinical symptoms. Furthermore, allergic and nonallergic rhinitis symptoms overlap substantially. In individuals with clinical symptoms and correlating positive allergy tests, however, improvement has been demonstrated with allergy medication, environmental control, and allergic desensitization (also

known as allergen immunotherapy). Allergy testing is imperfect, but useful.

In a 2009 survey sponsored by the Centers for Disease Control and Prevention, 11.8% self-reported “respiratory allergies”¹²; however, more than 4 times that number have a positive skin test. The Third National Health and Nutrition Survey examined the prevalence of positive skin-prick test (SPT) for a screen of inhalant allergens. It found that 53.9% of the 10,508 who were randomly tested had at least one positive SPT.²⁵ This disparity between clinical symptoms and skin testing is also displayed in a Swiss study. In the 1998 Swiss Study of Air Pollution and Lung Diseases (SAPALDIA), clinical allergic rhinitis was determined by surveying and testing 8329 randomly selected adults. They were asked, “In the past 12 months, did you suffer from allergic rhinitis, including hay fever?” or “Did you experience a runny or stuffy nose, the urge to sneeze, or itchy or watery eyes related to common allergen exposure?” The positive predictive power of an SPT predicting clinical allergic rhinitis in this study was 48.7%.²⁶ The following sections discuss the different types of allergy testing, but it is important to recognize that there is a significant amount of clinical judgment in deciding whom to test. The previously mentioned studies suggest that allergy testing should not be used as a screening test independent of clinical symptoms, as there is a significant false positive rate.

Allergy testing is helpful in determining to what patients might be allergic. There are different methods of allergy testing that are subdivided into challenge tests, skin tests, and IgE measurement.

Challenge Testing

Challenge testing is the exposure of an individual to an allergen and observing for an allergic reaction. Some of the first studies of allergic disease used conjunctival challenges with pollen.²⁷ For allergic rhinitis, it would involve placing allergen in the nose or placing the subject in a room with circulating allergen (an environmental exposure chamber). Challenge testing is used as the “gold standard” in food allergy²⁸ but is less agreed on in inhalant allergy testing. It might seem intuitive that challenge testing would be the gold standard for allergic rhinitis. If one placed ragweed pollen in the nose and the individual developed rhinorrhea and sniffing, then the person would be allergic to ragweed; however, challenge testing for inhalant allergens tends to exceed amounts encountered in natural exposures. Also, a single challenge may elicit a different response than a seasonal exposure. Perhaps for these reasons, challenge

testing has not correlated optimally with clinical symptoms.³ Additionally, challenge testing is difficult to standardize and suffers from subjective responses.²⁹ As such, challenge testing is relegated to research for allergic rhinitis.

Skin Testing

Skin testing is the most widely used testing for allergies in the United States. An extract of an inhaled allergen is placed beneath the skin's surface via scratch, prick, or intradermal injection. If mast cells in the skin degranulate after allergen exposure, histamine and other mediators produce a wheal (local edema) and flare (erythema). The skin reaction is compared with positive (histamine) and negative (diluent) controls and graded (or read) by the tester. Skin-testing results may vary by technique, area of the body, test interpreter, medications, and allergen extract, but serve as a useful and effective tool for assessing the risk of allergy.³⁰ A small risk of triggering a systemic allergic reaction is incurred with all types of skin testing.²¹

Scratch testing is not currently recommended, as reproducible results are difficult to obtain.³ Prick testing uses single-prick or multiprnick devices to place the extract in the epidermis. Prick tests can be reproduced with practice and are favored for their safety, cost, and reasonable correlation to clinical disease. Intradermal tests use a needle to raise a small wheal and observe the wheal for growth and erythema. Single intradermal tests use more extract than prick tests alone and may generate a response when the prick test is negative. Studies have questioned if intradermal tests add any clinical value when the prick test is negative.³¹ Intradermal tests using different dilutions of extract will show increasingly large wheals and flares in a sensitive subject.³⁰ Intradermal dilutional testing is used for antigen standardization and venom allergy testing, and was once widespread among otolaryngologists. As there is no agreed on gold standard for inhalant allergy testing, there is controversy about the best way to test for allergy. The most controversy surrounds using relatively high amounts of allergen applied intradermally.

slgE Testing

IgE is the molecule produced by plasma cells that recognizes the allergen and serves as the allergen receptor that triggers degranulation of mast cells and basophils. The plasma cells of the allergic individual constantly produce cloned specific IgE (slgE) that is both bound on cells that express IgE receptors and free in the circulation. Free

slgE can be sampled in plasma or sera and is measured by allowing it to bind to an allergen (such as ragweed pollen) adhered to a matrix. The patient's bound IgE is tagged with a labeled anti-human IgE, which is then measured. This technique also allows the amount of slgE to be indirectly quantified. In theory, any person with an IgE-mediated allergy would necessarily produce slgE to that allergen; however, technical issues exist.³²

slgE testing has undergone several evolutions of technology and is currently judged to be slightly less sensitive than skin-prick testing. Advantages include that multiple tests can be run off a single blood draw, results are not suppressed with anti-histamine use, and interpretation is not subjective.

Higher slgE levels correlate with stronger skin-test reactions, and lower slgE levels correlate with weaker skin reactions. Some evidence suggests that the higher the slgE level, the more likely it is that the allergy is clinically present. A clear example of this relationship is in food allergy testing. In IgE mediated food allergy, slgE levels correlate with the probability that the blinded food challenge will be positive.³³ Surprisingly, the degree of skin reactivity and level of slgE have not correlated well with the severity of clinical symptoms.³⁴ The concept that strongly positive test results predict a higher probability of clinical allergy but not greater symptom severity is somewhat counterintuitive.

Total IgE Testing

Total IgE can also be measured and again it is intuitive that allergic individuals would have high levels of total IgE. However, total IgE has not been found to be as helpful as slgE in determining who is allergic and who is not. This is partly because other inflammatory conditions, such as asthma, chronic eosinophilic sinusitis, and smoking, are associated with high total IgE. Also, many patients with allergic rhinitis defined by positive tests and history have total IgE in the normal range.^{32,35}

Testing Summary

Allergic rhinitis is a clinical diagnosis, not a test result. Testing is useful in separating allergic rhinitis and nonallergic rhinitis but loses specificity when applied independent of clinical impression. The accuracy of allergy testing also varies among different allergens. For some allergens, the extracts used in testing are standardized and the specific protein epitopes that bind to the IgE are shared among most allergic individuals. Cat and ragweed are examples of standardized extracts with a prominent "major allergens"; however, other

allergens, especially molds, are not standardized and have multiple allergenic epitopes but no “major allergen.” Most allergenic sources have multiple allergens and a “major allergen” is determined when more than 50% of the sensitized population reacts to that particular allergen.

TREATMENT OF ALLERGIC RHINITIS

Treatment of allergic rhinitis is traditionally divided into 3 categories: pharmacotherapy, environmental control, and immunotherapy (or desensitization). Because this article targets surgeons, we also discuss the role of surgical interventions. As pharmacotherapy is covered in depth in the article “Pharmacotherapy” by Chaaban and Corey elsewhere in this issue, the discussion in this article is cursory.

Pharmacotherapy

Pharmacologic management of allergic rhinitis is used in most patients, and billions of dollars are spent in allergic prescription and over-the-counter medications each year.¹ A brief comment on selecting the different categories of allergy medications is summarized in the following paragraphs.

Systemic steroids are highly effective at decreasing the inflammation from allergic rhinitis but carry a risk and side-effect profile that is difficult to justify when other options are available. For severe intermittent allergic rhinitis that is otherwise not well controlled, however, a short course may be reasonable.

Intranasal corticosteroids (INCSs), aside from systemic steroids, are the single most effective class of medical therapy for allergic rhinitis. There is little difference in clinical efficacy between available INCSs; however, differences in price, taste, and systemic effects exist. Local irritation, especially ulceration and bleeding from the nasal septum, is a common side effect with persistent use. Educating patients to spray parallel to the turbinates and to not angle the bottle toward the septum seems helpful in reducing local irritation in the author’s experience. Although intermittent use has demonstrated efficacy, maximal efficacy of symptom control occurs after 7 to 10 days of use. As such, INCSs are perhaps not the best choice for intermittent use or as a rescue medication.¹

Antihistamines are divided into first and second generations, with sedation being much less in the second-generation medications. Sedation with first-generation antihistamines can last into the following day and impairs work productivity, school performance, and driving. First-generation

antihistamines do not have a rational role in the management of allergic rhinitis for most patients. Antihistamines help with rhinorrhea, itching, and sneezing, but are similar to placebo for nasal congestion. Antihistamines are often paired with decongestants for this reason. Antihistamines are receptor blockers and are probably more effective if taken before a known allergen exposure, but they also work relatively quickly and are practical as a rescue medication. Cetirizine,³⁶ fexofenadine,³⁷ and levocetirizine³⁸ provide greater symptom control than loratidine dosed at 10 mg in environmental exposure unit studies. Intranasal antihistamines also work well and have an additive effect when used with INCSs; however, taste and sedation are issues for some patients.³⁹

Leukotriene modifiers are effective for allergic rhinitis, control symptoms comparable with loratidine, and have an added effect when combined with antihistamines, approaching INCSs.⁴⁰ Generally, leukotriene modifiers are used more in patients with allergic rhinitis and asthma.

Cromolyn requires frequent dosing and has mild efficacy, but has excellent safety.¹

Decongestants are effective with nasal congestion and rhinorrhea but can have significant side effects, including sleep disruption and hypertension. They are best fitted for intermittent allergic rhinitis or as a rescue medication in young adults without hypertension.¹

Environmental Control

Environmental avoidance is recommended in multiple position papers, but has poor support from controlled studies in consistently improving symptoms. It is helpful to classify the studies into 3 groups.

Allergic subjects are physically removed from allergenic source

Mostly looking at allergic asthma, studies have shown that moving sensitized patients to an allergen-free location improves symptoms. In one study, schoolchildren with asthma and allergic to dust mites were enrolled in a school located at a high altitude in the Alps where dust mites could not survive. They had significant improvement in their asthma.⁴¹ Similar improvements in asthma are sometimes seen when patients are admitted into a hospital. Although effective, this strategy is frequently impractical.

Environmental control techniques with allergen measurement as the outcome

Multiple studies will measure the amount of allergen in the air or grams of dust and then apply an intervention, such as dust-mite covers.

Postintervention measurements are compared for statistical significance. By this standard, many interventions, including dust-mite covers,⁴² acaricides,^{43,44} vacuum cleaning,⁴⁵ HEPA filters,⁴⁶ washing linens,⁴⁷ removing carpets,⁴⁸ and washing pets⁴⁹ are effective. Of note, washing cats reduces levels for only a single day. Despite reduction of measured allergens, the clinical implication of this is less certain.

Environmental control techniques with allergy symptom outcomes

Improvement of symptoms or decreased medication use is much more difficult to demonstrate. An example of this type of study would be a randomized blinded placebo-controlled trial published in the *New England Journal of Medicine* that looked at the effect of dust-mite covers on allergic rhinitis in 232 dust mite-sensitized patients. The investigators showed no difference in their clinical outcome measures.⁵⁰ In many cases, the reduction of allergen seems to be insufficient to benefit the allergic patients. One of the interesting features of allergic reactions is how a small exposure can trigger symptoms. Studies that have used rigorous environmental control with multiple interventions have shown some success.⁵¹

In allergic rhinitis, patients wishing to use environmental controls as a strategy should commit to multimodalities and stringent interventions (removing carpets or pets), or expect modest results.⁵¹ When paired with medical therapy, environmental controls and avoidance can result in subjective improvement in some patients. This topic is also discussed elsewhere in this issue.

Surgery as an Adjunct for Allergic Rhinitis?

Nasal congestion is a primary symptom of allergic rhinitis and is likely the symptom most responsible for the impairment of quality of life and sleep in those with allergic rhinitis.¹ Hypertrophy of the inferior turbinate is partially responsible for the nasal obstruction. A recent study randomized 55 patients with allergic rhinitis to radiofrequency reduction of the inferior turbinates or intranasal mometasone spray. At 1 year, turbinate reduction provided significantly better decreased nasal resistance, significantly improved congestion subjectively assessed by visual analog scale, and similar improvements in a validated quality-of-life instrument as compared with intranasal mometasone. The turbinate reduction group was significantly better in the domains of sleep and nasal symptoms.⁵² Two other studies support these findings. Mori and colleagues⁵³ reported benefit in quality of life in patients with perennial allergic rhinitis 5 years after submucosal reduction of the

turbinates. Lin and colleagues⁵⁴ reported improved symptoms in 108 subjects with allergic rhinitis refractory to medical therapy after radiofrequency turbinate reduction assessed 1 year after surgery. Take together, these 3 studies provide data that support considering turbinate reduction for allergic rhinitis in selected patients. Surgery may also have a role when allergic rhinitis coexists with nasal valve collapse, deviated septum, nasal polyps, adenoid hypertrophy, or chronic sinusitis, in the author's opinion.

Immunotherapy

Although pharmacotherapy, environmental avoidance, and possibly surgery may help manage allergic rhinitis, only immunotherapy has the potential to permanently decrease or resolve the underlying allergic inflammation. Allergen immunotherapy is the regular exposure of the sensitized patient to the allergen(s) to which the patients is sensitized so as to promote tolerance.⁵⁵ Inhalant allergen immunotherapy is classified by the route the allergen extract is delivered. Immunotherapy can be provided subcutaneously (shots), sublingually, intralymphatically,⁵⁶ orally, or nasally. This article discusses only subcutaneous immunotherapy (SCIT) and sublingual immunotherapy (SLIT). The mechanism of immunotherapy is not known precisely, but likely depends on changes in T-helper lymphocytes down-regulating inflammation and shifting the inflammatory mediator profile toward nonallergic.

SCIT

Subcutaneous immunotherapy, or allergy desensitization shots, involves injecting the allergens to which the patient is sensitized (allergic) into the subcutaneous tissue (usually of the arm). There are 2 phases of SCIT: escalation and maintenance. During escalation, the patient is started at a sufficiently dilute dose to be safe and each subsequent shot contains a higher amount of allergen. Through serial shots provided once or twice a week, the amount of allergen is increased several thousand fold. Once a tolerated dose in the effective range is established, maintenance therapy involves giving the same dose at regular intervals. Gradually the time between shots is increased to monthly. The duration of maintenance therapy is generally 3 to 5 years and most patients can discontinue and retain the clinical benefit, although there is significant individual variation.⁵⁵

Randomized controlled trials and meta-analyses have supported efficacy for SCIT in single-antigen studies⁵⁷; however, most patients with allergic rhinitis have multiple sensitivities and are desensitized using combined allergens. SCIT

exposes allergic patients to higher than natural exposures of allergen and each shot poses the risk of triggering anaphylaxis. Time, expense, and anaphylactic risk of immunotherapy need to be considered in selecting and counseling patients. Immunotherapy is reserved for moderate and severe allergic rhinitis not controlled easily with other measures.

SLIT

Sublingual immunotherapy is performed by placing drops of allergen extract under the tongue. Holding the extract under the tongue seems to allow the allergens to be taken up into the regional lymphatics. Multiple studies using different allergens, techniques (spit or swallow), frequencies, durations, and doses have been published, and meta-analyses have suggested effectiveness, although none of the variables is agreed on.⁵⁸ Initial industry-sponsored studies looking at sublingual therapy for grass allergy have also shown efficacy and safety,^{59,60} although SLIT is not approved by the Food and Drug Administration. While initial studies showed excellent safety, some reports of systemic reactions have been presented, especially after the first dose.⁶¹ Many sublingual protocols recommend daily drops given at home, and controversy exists as to level of safety expected for home therapy treating a nonlethal disease. Interestingly, systemic allergic reactions treated with epinephrine also appear in the placebo arms of some studies. The combination of safety, efficacy, and convenience make SLIT a compelling option. In the author's opinion, some of the controversy over SLIT may be biased by its potential socioeconomic impact.

SUMMARY

Facial plastic surgeons should develop expertise in nasal anatomy and pathophysiology to address functional and aesthetic consequences of nasal surgery. One area where allergic rhinitis may impact plastic surgery outcomes is aesthetic rhinoplasty. Aesthetic rhinoplasty can alter nasal function by narrowing the nasal valve. Nasal obstruction is more likely in patients with allergic rhinitis, as they have intermittent or persistent turbinate swelling. Identifying and managing allergic rhinitis in patients considering cosmetic nasal surgery should lead to better surgical outcomes and may inform decisions on the functional consequences of nasal surgery. Allergic rhinitis is primarily a clinical diagnosis and allergy testing is complementary. Allergy testing alone should not be relied on for screening, as the false positive rate is likely unacceptable. Allergic rhinitis

is manageable in most individuals through choices of pharmacotherapy, environmental control, and allergen immunotherapy. There may be a role for turbinate reduction, nasal valve surgery, and septoplasty in selected patients with allergic rhinitis refractory to nonsurgical management. The diagnosis and management of allergic rhinitis should assist in better selection, counseling, and outcomes for patients considering aesthetic nasal surgery.

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