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OFFICE MANAGEMENT: ALLERGY AND IMMUNOLOGY

Richard D. deShazo, Speciality Editor

Office-Based Management of Allergic Rhinitis in Adults

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ABSTRACT

Allergic rhinitis (AR), the most common form of rhinitis, affects 10% to 30% of adults. However, the prevalence of AR actually may be underestimated. Rhinitis is caused by allergic, nonallergic, infectious, hormonal, occupational, and other factors. Proper management of AR requires that the correct diagnosis be made; the clinical history and physical examination are key. Controlled studies support the utility of several available treatments that produce fewer symptoms, improve quality of life, and prevent comorbidities. © 2007 Elsevier Inc. All rights reserved.

KEYWORDS: Adult; Allergic rhinitis; Management; Outpatient; Perennial; Seasonal

Allergic rhinitis (AR), the most common form of rhinitis, affects 10% to 30% of adults and results in millions of annual office visits for its symptoms, sinus disease, and otitis media with effusion.¹ Symptoms include rhinorrhea, nasal obstruction, nasal itching, and sneezing, which are reversible spontaneously or under treatment.² The majority of patients with asthma have AR. Rhinitis symptoms in patients with asthma are associated with worse asthma outcomes.² AR has a significant impact on the quality of life (QOL) of patients.¹ Sleep-disturbed breathing is commonly associated with AR and causes significant impairment in work and school.¹

Traditionally, AR has been subdivided into seasonal AR (SAR) and perennial AR (PAR). SAR symptoms usually appear during a specific season in which aeroallergens, such as tree and grass pollen in the spring and summer and weed pollens in the fall, are present in outdoor air. PAR symptoms are present year-round and are triggered by dust mite, animal dander, indoor molds, and cockroaches.³ A new classification for AR based on the persistence and severity of symptoms has been proposed.²

DIAGNOSIS OF ALLERGIC RHINITIS IN THE ADULT

Rhinitis may be caused by allergic, nonallergic, infectious, hormonal, occupational, and other factors. The presence of

allergic triggers in rhinitis is often unrecognized by both physicians and patients.¹ Clinical history is essential for an accurate diagnosis of AR, assessment of severity, and response to treatment (Table 1).

Examination of the nose can be accomplished with a nasal speculum with appropriate lighting or an otoscope with a nasal adapter. The typical nasal mucosa of the allergic patient appears pale and swollen, with a bluish-gray appearance when the mucosal edema is severe. The mucosa is usually reddened in acute infections and with overuse of topical medications. However, mucosal appearance may not distinguish between allergic and non-AR, because non-AR also may present with mucosal pallor, edema, or hyperemia. Examination of the nasal cavity may identify anatomic abnormalities such as septal deviation, polyps, foreign bodies, or masses that contribute to symptoms.

With chronic or severe AR, a transverse nasal crease is often seen across the bridge of the nose, particularly in children, as a result of rubbing of the nose to relieve itching. Accentuated folds below the margin of the inferior eyelid, referred to as Dennie-Morgan lines, might be present. Infraorbital dark skin discoloration, termed "allergic shiners," can be present with any type of nasal obstruction. The palpebral conjunctivae may be injected with a watery discharge and puffiness of the eyelids. The tympanic membranes should be examined for evidence of associated middle-ear disease from eustachian tube involvement. Patients also should be evaluated to exclude coexistent purulent sinusitis, asthma, and atopic dermatitis.

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Table 1 Important Historical Points in the Evaluation of Rhinitis

- Symptoms: magnitude, duration, timing in relation to exposure (ie, early and/or late-phase allergic reactions), effects on daily living
- Triggers/seasonality (ie, spring: tree, grass, and pollen allergy; fall: weed pollen allergy)
- Environment, including home, job, and school or day care for children
- Impairment of school/work performance, interference with leisure activities, and any sleep disturbances
- History of other allergic symptoms (eg, asthma, conjunctivitis, eczema)
- Medical history, including trauma
- Feeding history in young children
- Past treatment experience
- Current treatment response
- Family history, including allergic diseases

DIFFERENTIAL DIAGNOSIS

Because approximately 50% of patients with rhinitis do not have AR, other potential causes also must be excluded.¹ Table 2 highlights a list of alternate causes to keep in mind.⁴

Table 2 Differential Diagnosis of Rhinitis

Allergic	Conditions that Mimic Symptoms of Rhinitis
Seasonal/Intermittent	Structural/Mechanical
Perennial/Persistent	Deviated nasal septum
Occupational	Polyps
Non-allergic	Foreign bodies
Infectious	Adenoidal hypertrophy
NARES	Hypertrophic turbinates
Perennial non-allergic (vasomotor)	
Other	Choanal atresia
Ciliary dyskinesia syndrome	Tumors
Atrophic	Inflammatory/Immunologic
Hormonal	Sarcoidosis
Hypothyroidism	Wegener's granulomatosis
Pregnancy	Sjögren syndrome
Menstrual cycle	Systemic lupus erythematosus
Exercise	Cerebrospinal fluid rhinorrhea
Drug-induced	
Aspirin/NSAIDs	
Oral antihypertensives	
Rhinitis medicamentosa	
Oral contraceptives	
Reflex-induced	
Gustatory	
Irritant	
Posture reflexes	
Nasal cycle	
Emotional factors	

NARES = nonallergic rhinitis-eosinophilia syndrome; NSAID = nonsteroidal anti-inflammatory drug.

Table 3 Guidelines for the Treatment of Allergic Rhinitis

- Identify the allergen by history or tests
- Allergen avoidance
- Begin an intranasal corticosteroid, oral second-generation H₁ antihistamine, nasal antihistamine, or combination; an oral or nasal decongestant may be used short term
- For resistant nasal symptoms add a leukotriene receptor antagonist; for resistant ocular symptoms add a mast cell stabilizer or an NSAID
- Consider immunotherapy if relief with medication is inadequate or to prevent further progression of allergic disease

NSAID = nonsteroidal anti-inflammatory drug.

DIAGNOSTIC EVALUATION

The diagnosis of AR can usually be made from the history and physical examination. Allergy testing can be performed to identify which allergens are responsible for the symptoms. There are 2 methods used to determine the presence of immunoglobulin (Ig)-E antibodies to specific allergens: skin testing, and radioallergosorbent (RAST) testing.

Skin testing is typically performed with a set of allergens relative to the patient's environment and clinical history.⁵ Skin testing involves introduction of the allergen extract into the skin by "prick" or intradermally. Histamine is used as a positive control, and saline is used as a negative control. The wheal and flare response to a specific allergen is then compared with the controls. To avoid false-negative tests, antihistamines should be withheld for 36 to 48 hours.³ In vitro measurement of allergen-specific IgE also can be performed using the RAST test. Skin testing has greater sensitivity, is less expensive, and provides immediate results, whereas RAST is limited in the selection of allergens but has the advantage of not being affected by antihistamines. The demonstration of IgE-specific antibodies alone does not establish that the patient will have an adverse response on exposure to the allergen because sensitization can occur without reaction to inhalation. Thus, results must be interpreted along with the clinical picture before a diagnosis can be made.

MANAGEMENT

An effective treatment strategy (Table 3) should begin with allergen avoidance when possible. Pharmacologic management consists of several classes of medications, including oral and intranasal H₁-antihistamines, intranasal corticosteroids, leukotriene modifiers, mast cell stabilizers, and decongestants. Ophthalmologic preparations, such as mast cell-stabilizers, antihistamines, antihistamine/mast cell-stabilizer combination agents, and nonsteroidal anti-inflammatory drugs, can be used in patients with ocular involvement. Allergen-specific immunotherapy in selected patients is the only treatment modality that is disease modifying.³

ENVIRONMENTAL CONTROL

Avoidance of known allergic triggers is often impractical.¹ However, decreased exposure to indoor allergens can result in improvement to a level that regular medications are not required in some patients. Allergen-avoidance measures are shown in Table 4.²

INTRANASAL CORTICOSTEROIDS

Intranasal corticosteroids are the first-line therapy for moderate–severe AR² (Table 5). Corticosteroids target the inflammatory mechanism of the early and late-phase allergic processes and are therefore effective in treating most symptoms of AR, including congestion, sneezing, rhinorrhea, and nasal pruritis.^{2,6} A meta-analysis compared the effects of intranasal corticosteroids with oral antihistamines in the treatment of AR and found a clinically and statistically significant benefit to intranasal corticosteroids over oral antihistamines for nasal congestion and sneezing.^{5,7}

Adverse systemic side effects seem to be minimal in adults.⁶ Epistaxis, secondary to irritation of the nasal mu-

Table 5 Available Once-Daily Intranasal Corticosteroids	
	Usual Daily Adult Dose
Triamcinolone acetonide	
Aerosol	1-2 sprays/nostril daily
HFA	2-4 sprays/nostril daily
Budesonide	1-4 sprays/nostril daily
Fluticasone	2 sprays/nostril daily
Mometasone furoate	2 sprays/nostril daily
HFA = hydrofluoroalkane.	

cosa, is the most common adverse effect but usually diminishes over time.^{1,2} Therefore, patients should be instructed on the proper technique for administration (Figure). It also is important to routinely examine the nasal mucosa in patients using topical nasal steroids to look for signs of irritation and/or septal perforation.

Children and adults should receive the lowest possible dose of intranasal corticosteroid, and children should have routine height monitoring to observe for suppression of linear growth.¹ Some studies have suggested a link among posterior subcapsular cataracts, glaucoma, and intranasal corticosteroids.¹

ORAL AND INTRANASAL ANTIHISTAMINES

The older first-generation H₁-antihistamines, such as diphenhydramine and chlorpheniramine, are sedating antihistamines. These agents are effective in AR but cross the blood-brain barrier and are associated with sedation leading to impaired performance at home, work, and school.¹ With this high risk-to-benefit ratio, the first-generation H₁-antihistamines are not recommended.

The newer second-generation H₁-oral antihistamines have improved H₁-receptor selectivity, absent or decreased sedation, longer duration of action, and fewer adverse effects.⁸ They are recommended as first-line therapy for the treatment of mild–moderate AR² (Table 6). H₁-antihista-

Table 4 Recommendations for Allergen Avoidance	
Allergen	Avoidance Measures
Indoor	
Molds	● Use a bleaching agent on tile grout, sinks, shower walls, and garbage cans ● Avoid humidifiers ● Dehumidifiers in damp areas
Dust mites	● Bed linens should be washed in hot water (>130°F) once every other week ● Pillow and mattress encasings ● Removal of carpet from homes ● Treat carpet with acaricides and the use of vacuum cleaners with (HEPA) filters and double-thickness bags
Animal dander	● Furry pets should be removed from the home* ● Carpets and furniture cleaned ● Confine pet to a room with a HEPA or electrostatic air purifier
Cockroach	● Efforts should be made to prevent infestation
Outdoor	
Pollens	● Keep windows closed ● Use air conditioning with external vents closed ● Avoid window or attic fans ● Wear mask and/or goggles while mowing grass
HEPA = high-efficiency particulate air.	
*Cat allergen can remain in the home for up to 6 months after removal of the pet.	

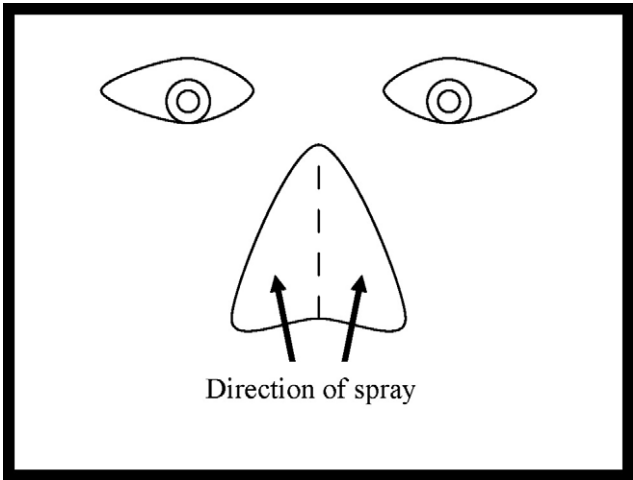


Figure Proper intranasal steroid technique.

Table 6 Available Second-Generation H₁-Antihistamines

	Usual Daily Adult Dose	Nonsedating
Oral		
Cetirizine	5-10 mg	No
Desloratadine	5 mg	Yes
Fexofenadine	60 mg twice daily; 120 mg or 180 mg daily	Yes
Loratadine	5-10 mg	Yes
Topical		
Azelastine	1-2 sprays nostrils/once or twice daily	No

mines are effective in the treatment of nasal sneezing and pruritis.² Clinical trials assessing QOL in patients with AR demonstrate that these drugs consistently improve QOL.

Topical second-generation H₁-antihistamines are similar in efficacy to oral antihistamines and also considered as first-line therapy for mild-moderate AR.^{1,2} Studies show that azelastine improves all symptoms in SAR and PAR, including ocular symptoms, and also reduces nasal congestion.^{1,2,9}

LEUKOTRIENE MODIFIERS

Leukotrienes seem to be important mediators of nasal allergic reactions, and their application to the nose induces nasal obstruction.² They are involved in both the early and the late-phase allergic response. A meta-analysis demonstrated that montelukast was modestly better than placebo, as effective as antihistamines, but less effective than nasal corticosteroids in improving symptoms and QOL in patients with SAR.^{5,10} Montelukast has been generally accepted as an adjunct in the treatment of AR as opposed to monotherapy.

MAST CELL STABILIZERS

Mast cell stabilizers inhibit mast cell degranulation and thus inhibit the release of histamine and other mediators of the early phase of allergic inflammation. Cromolyn sodium, which is available over-the-counter, is generally not as effective as antihistamines or intranasal corticosteroids in reducing symptoms.² Cromolyn is likely to be more effective when administered just before contact with an allergen.² The dosing interval of 4 times per day make this a less attractive option.

Ophthalmic mast cell stabilizers, ocular antihistamines, mast cell-stabilizing/antihistamine agents, and the nonsteroidal anti-inflammatory drug ketorolac (Table 7) have all been effective when used topically for the treatment of ocular symptoms associated with allergic conjunctivitis that often accompany AR.⁵

DECONGESTANTS

Decongestants reduce nasal congestion by activating α -adrenergic receptors on the nasal vasculature leading to vaso-

constriction.² Decongestants have not been shown to be effective with other symptoms of AR. A recent study showed that the combination of pseudoephedrine and an antihistamine was significantly more effective in reducing total nasal symptoms than either agent alone.¹¹ Caution must be taken when prescribing decongestants to patients with hypertension, diabetes, prostate enlargement, or hyperthyroidism, or patients taking monoamine oxidase inhibitors.

Topical decongestants, oxymetazoline and phenylephrine, are also available over-the-counter. These medications can be effective with nasal congestion associated with AR. Rebound nasal congestion, termed "rhinitis medicamentosa," can occur if these medications are used for more than 3 to 5 days.

IMMUNOTHERAPY

Subcutaneous immunotherapy (SCIT) is indicated for the treatment of AR, and expert guidelines recommend its use for patients who continue to have moderate-severe symptoms despite therapy, have intolerable adverse effects with pharmacotherapy, do not want to be on pharmacotherapy, and have coexisting conditions such as asthma.^{2,5} SCIT consists of administration of increasing amounts of the culprit antigen over several months until a maintenance dose is reached. The maintenance dose is usually given at intervals ranging from 2 to 6 weeks.⁵ Drawbacks to SCIT include the frequent injection schedule and the requirement for the injections to be done in the office with a 20- to 30-minute SCIT observation period.

The most common adverse effects associated with SCIT are local injection site swelling and erythema. However, systemic reactions, including anaphylaxis, have occurred but are less common.¹²

The response rate to therapy in carefully selected patients is good with trials reporting a two-thirds reduction in symptoms.¹³ Other studies have shown persistent effects after the SCIT was stopped.⁷ SCIT is the only therapy that can alter the natural course of the disease.³

Table 7 Available Ocular Agents

	Usual Daily Adult Dose
Mast cell stabilizers	
Cromolyn sodium	1-2 drops OS/OD 4-6 times daily
Pemirolast	1-2 drops OS/OD 4 times daily
Topical antihistamines	
Emedastine	1 drop OS/OD 4 times daily
Multiple-action agents	
Olopatadine	1-2 drops OS/OD twice daily
Azelastine	1 drop OS/OD twice daily
Epinephrine	1 drop OS/OD twice daily
Ketotifen	1 drop OS/OD twice daily

OS = oculus sinister; OD = oculus dexter.

Table 8 Managing Allergic Rhinitis During Pregnancy

- Avoid known allergens
- Ocular symptoms can be managed with topical cromolyn supplemented with loratadine or cetirizine (both pregnancy category B) as needed
- Mild intermittent rhinitis symptoms can be treated with loratadine or cetirizine
- Addition of intranasal budesonide (pregnancy category B) used intermittently for moderate-severe symptoms
- Intranasal cromolyn can be used for mild, persistent rhinitis symptoms supplemented with loratadine or cetirizine with the addition of intranasal budesonide used regularly for moderate-severe symptoms
- Immunotherapy is not recommended unless the patient was on this before pregnancy with good response. The dosage should not be escalated during pregnancy.

MANAGING PREGNANT PATIENTS

Managing AR in pregnancy and lactation can pose a challenge to the treating physician, mainly because of the medical-legal aspects. However, there are suitable pharmacologic agents available for use in these patients. Recent recommendations are summarized in [Table 8](#).

REFERRAL TO AN ALLERGIST

Referral to an allergist is indicated for those patients with a questionable diagnosis or poor response to medications, if systemic steroids are needed to control symptoms, or there are other comorbid conditions such as asthma or if immunotherapy is considered.

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