
Seasonal Allergic Rhinoconjunctivitis

Allergic rhinoconjunctivitis is a common condition. Most patients can achieve good symptom control through allergen avoidance and pharmacotherapy with non-sedating antihistamines and topical agents, particularly intranasal corticosteroids.

By Karen Binkley, BSc, MD, FRCPC

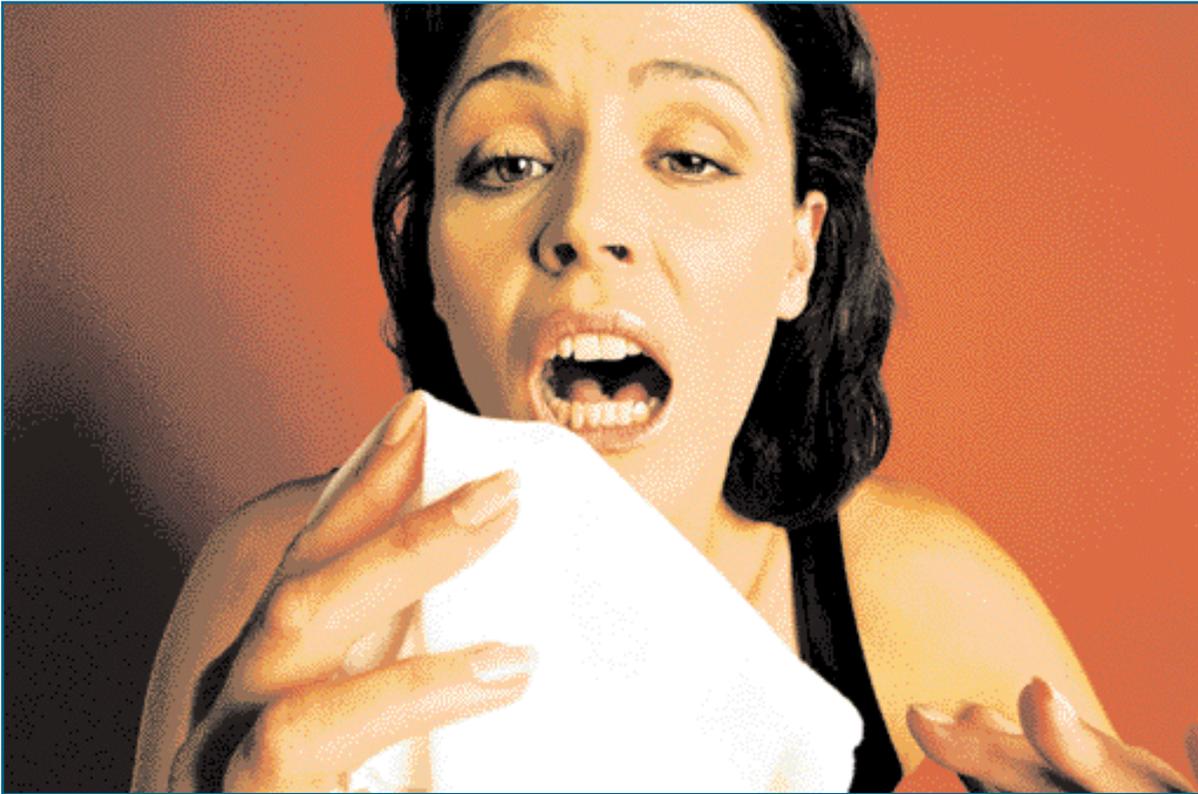
Seasonal allergic rhinitis affects approximately 15% of the population. It can cause significant deterioration in quality of life for afflicted individuals. Occasionally, more serious complications, such as acute sinusitis, can arise if early symptoms are not treated properly. An understanding of the underlying process and available treatments forms the basis for effective intervention.

Sensitization

In an individual genetically predisposed to mount immunoglobulin E (IgE) responses, exposure to airborne allergens leads to the

production of pollen-specific IgE antibodies. These antibodies attach to high affinity receptors located on the surface of mast cells and basophils, which are in turn located at mucosal surfaces. When an airborne allergen comes in contact with IgE on the mast cells and basophils at the mucosal surface, adjacent IgE molecules are cross-linked, causing a signal to be transmitted across the mast cell membrane. This culminates in mast cell degranulation, starting a cascade of mediator release.

Histamine is a prominent mediator, but others include prostaglandins, leukotrienes, platelet activating factor, interleukins and many others. Together they cause the famil-



ilar symptoms of itchy, red, watery eyes, as well as sneezing, rhinorrhea and nasal congestion.

Many of the mediators released by the mast cells act as chemoattractant factors. A gradient of these factors diffuses away from the sight of allergen contact, resulting in an influx of inflammatory cells—particularly

eosinophils—into the area of allergen contact. As these cells invade the area of allergen contact, they are stimulated to release additional mediators of inflammation. This constitutes the so-called “late-phase IgE reaction,” which corresponds to clinical allergy.

Diagnosis

Typical symptoms of rhinoconjunctivitis that recur in the same season year after year strongly support the diagnosis of seasonal allergies. Signs and symptoms of other atopic conditions, including asthma, eczema and food allergy, also may be present. There may be a family history of atopy.

A knowledge of the pollen patterns in the geographic area, combined with an accurate



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history, can help identify relevant culprit aero-allergens. In Southern Ontario, for example, trees begin pollinating in mid-to-late April and finish by early June. Grass pollen begins at the end of May and ends in early July. Leaf moulds peak in July and August, and ragweed season begins in early-to-mid August and continues until the first frost.

Allergy Skin Testing

Allergy skin prick testing can confirm the diagnosis. A small amount of extract of the relevant pollen protein is introduced into the skin. If pollen-specific IgE is present, mast cell mediator release occurs and is assessed indirectly by the formation of a wheal and flare reaction at the site.

The results of allergy skin testing should always be correlated with the patient's history. For example, a positive ragweed skin test does not account for perennial symptoms, which are probably non-allergic. A positive skin test in the absence of symptoms usually does not indicate the need for treatment.

Treatment

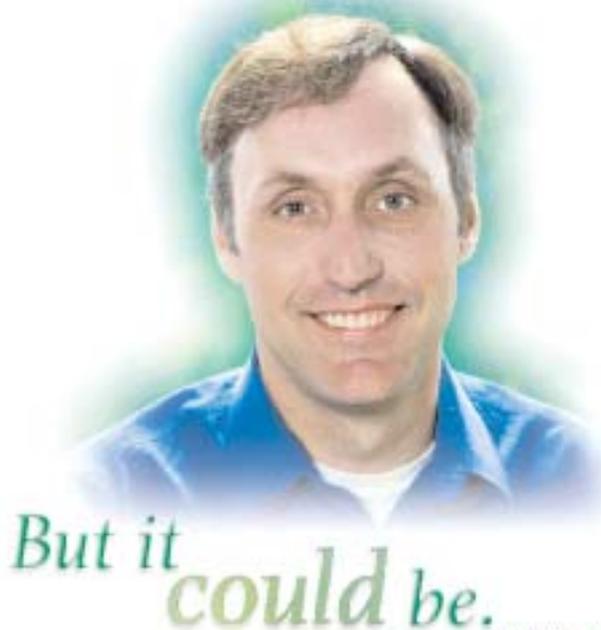
Avoidance of the offending allergen(s) is the first step in all treatment strategies.¹ Once the allergen(s) are identified by history and appropriate allergy testing, specific avoidance strategies are targeted to the key seasons. Keeping doors and windows in the home closed will significantly reduce pollen counts indoors and result in symptomatic improvement. Air conditioners can be used for cooling purposes, but vents to the outdoors should be closed to avoid bringing in the pollen-laden outdoor air. These measures

need only be taken while the offending pollen is "in season."

The next recommended step in treatment is pharmacotherapy (Table 1). Topical nasal steroids are the most important component of medical therapy. These should be started just prior to, or early in, the relevant season and used on a regular basis throughout.² Topical nasal corticosteroids are rapidly efficacious, effective, well tolerated and safe. They offer significant relief in the vast majority of patients.

Adequate control of mucosal inflammation promotes adequate drainage of nasal secretion and may prevent the development of acute sinusitis. At recommended doses, there is no significant evidence of adrenal suppression. Mucosal biopsies performed after years of continuous therapy have shown

COPD
is *seldom* diagnosed
before the sixth decade.



Allergies



no evidence of nasal atrophy. Epistaxis is a common side effect, but is rarely severe. Septal perforation also is a rare event. Contraindications for the use of these agents include known hypersensitivity to their components and untreated fungal infections.

Nasal cromoglycate, which is a mast cell stabilizing agent, is significantly less potent as an anti-inflammatory agent. It can be effective in mild cases.

Nasal antihistamine preparations are available. They block the action of histamine, but do not affect the other mediators. While they offer some relief, they are less effective than intranasal corticosteroids.

Conjunctival symptoms, if present, can be treated with either topical mast cell stabilizing agents, antihistamines or medica-

COPD *The evidence*



at 40-50



at 50-55



at 55-60



tions that are both mast cell stabilizing agents and antihistamines. Topical corticosteroids are generally avoided in the eye because of risk of cataract, and increased intraocular pressure.

Systemic treatment with non-sedating antihistamines can be used as sole therapy for mild cases or can be added to the above regime.

Topical nasal decongestants should be avoided. Prolonged use leads to the development of rhinitis medicamentosa, with rebound nasal congestion.

Systemic decongestants, available alone or in combination with antihistamines, can provide additional relief, but may cause anxiety, insomnia and elevated blood pressure. Generally, they are not desirable for long-term use.

Immunotherapy can be effective for seasonal allergic rhinoconjunctivitis.^{3,4} It is generally reserved for patients who have tried an adequate trial of allergen avoidance and pharmacotherapy. Fewer than 10% of patients with allergic rhinitis require immunotherapy.⁵ Used properly in appropriately selected cases, about 70% of patients note some relief with immunotherapy. Occasionally, relief can be dramatic, but more often it is incomplete and therapy with medications is still required.

Allergen immunotherapy is reserved for patients who fail an adequate trial of allergen avoidance and pharmacotherapy.

can be there *before 50.*

Diagnose Early. Treat with anticholinergic foundation therapy.²



at 60-70

Atrovent®

(ipratropium bromide)
Bronchodilator



Combivent®

(ipratropium bromide and salbutamol sulfate)
Bronchodilator



Or, to simplify treatment when a short-acting β₂-agonist should be added^{1†}

Atrovent inhalation aerosol is indicated for the maintenance therapy of responsive cases of chronic reversible airways obstruction, such as chronic bronchitis and asthma.

Combivent inhalation aerosol is indicated for the treatment of bronchospasm associated with chronic obstructive pulmonary disease (COPD).

The most common side effects of Atrovent were dry mouth in throat (9.4%), headache (2.9%), bad taste (3.8%) and palpitations (2.1%) (N=685).

The most common side effects of Combivent were headache (1.1%), bronchitis (0.1%) and cough (1.4%) (N=338).

^{††} Excess patient is well controlled on each agent separately and that doses are equivalent.

1. Guidelines for the Treatment of Chronic Obstructive Pulmonary Disease (COPD). In: *Statistics (BR), Canadian Respiratory Review (Time)*

2. Chapman KR. *Am J Med* 1996; 100 (suppl 1): 1A-35 - 1A-45

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Table 1

TREATMENT

Eye Drops

1. Antihistamines
 - Levocabastine
2. Mast cell stabilizers
 - Lodoxamide
 - Nedocromil
 - Cromoglycate
3. Combination antihistamine and mast cell stabilizer
 - Olopatadine

Nasal Preparations

1. Antihistamines
 - Levocabastine
2. Mast cell stabilizers
 - Cromoglycate
3. Corticosteroids
 - Beclomethasone
 - Budesonide
 - Flunisolide
 - Fluticasone
 - Mometasone
 - Triamcinolone
4. Nasal decongestants (not recommended; should not be used for more than 3-4 days at a time)
 - Oxymetazoline
 - Phenylephrine
 - Xylometazoline

Desensitization should be limited to those cases where well-standardized commercially available allergens exist.

Immunotherapy should be avoided in: patients with poorly controlled asthma (forced expiratory volume in one second [FEV₁] less than 70%); patients taking beta blockers and/or angiotensin-converting enzyme (ACE) inhibitors (which would interfere with the treatment of a systemic reaction should one occur); or any other patient who might tolerate a systemic allergic reaction poorly.

Future Therapies

In the future, a number of promising therapies may be useful. Monoclonal antibodies directed against IgE show benefit in the treatment of asthma, and would be expected to give significant relief for patients with allergic rhinoconjunctivitis. The high cost of this therapy, however, might limit its use.

Deoxyribonucleic acid (DNA)-based immunotherapy vaccines are being developed that may be more effective than conventional immunotherapy, with fewer side effects.

Conclusions

Allergic rhinoconjunctivitis is a common condition. Most patients can achieve good symptom control through allergen avoidance and pharmacotherapy with non-sedating antihistamines and topical agents, particularly intranasal corticosteroids. Allergen immunotherapy is reserved for patients who fail an adequate trial of allergen avoidance and pharmacotherapy. New therapies on the horizon offer hope to those few patients who do not achieve good symptom control with currently available treatments. **Dx**

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