Editor's message

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Allergic rhinitis remains an important disease, not because of mortality, but because of its frequency and morbidity. Few other diseases cause quality-of-life issues for the sufferers year after year and, in most cases, day after day, often lifelong. Daytime sedation, fatigue, poor sleep, decreased learning, decreased productivity have been well established as consequences of rhinitis. The expert opinion at present is that sleep-disordered breathing secondary to nasal congestion results in the daytime symptoms. When treating patients with allergic rhinitis, the healthcare provider needs to consider not only the symptom complex and which medication works best to control the symptoms, but also whether the medication would have a positive or negative effect on the daytime sedation and deceased productivity. Obviously, the treatment of choice is always avoidance, but when avoidance fails, medications are often necessary.

First-generation antihistamines (sedating antihistamines) control symptoms of rhinitis, with the exception of congestion; however, they worsen sleepiness and decrease alertness and ability to learn. Therefore, they are relatively contraindicated for the treatment of rhinitis. Second-generation antihistamines (low-sedating effect or nonsedating) are tolerated better, have fewer side effects, and do not decrease quality of life and productivity. When using second-generation antihistamines, it is important to note that they do not reduce nasal congestion and associated daytime somnolence. Intranasal corticosteroids (INCS) are not only effective for congestion but, with the exception of eye symptoms, they also seem to be more effective than antihistamines.

Nasal steroids have been shown to improve productivity, improve sleep, and improve daytime somnolence most likely secondary to the ability of INCS to decrease nasal congestion. No other mode of rhinitis therapy has the clinical effectiveness or the cost-effectiveness of INCS. All INCS are tolerated well and are beneficial, with few differences between them. To further explore their differences is beyond the scope of an editorial. Azelastine has been compared with nasal steroids with mixed results.

It appears that azelastine hydrochloride is effective in decreasing nasal congestion. The absorption across the nasal mucosa somewhat limits the use of azelastine because up to 12% to 20% of patients will experience sedation when using it. Because of the taste and sedating effect of azelastine, most clinicians limit azelastine to a third-line intervention for the treatment of rhinitis. When used, it is effective at reducing all the symptoms of rhinitis. Other alternative medications are cromolyn sodium, ipratropium bromide, and immunotherapy.

Cromolyn sodium is a weak anti-inflammatory agent and is limited by its need to be frequently dosed and its minimal benefit. The only instance when cromolyn appears to be indicated is for occasional predictable exposure. Starting cromolyn therapy before, during, and a short time after exposure may prevent symptoms. Ipratropium is only effective for rhinorrhea, and thus is rarely the medication of choice.

Immunotherapy (presently referred to as allergy vaccine therapy) is the only therapy that can be curative, or at least control symptoms even 3 or more years after stopping it. Allergy vaccine therapy can modulate the immune system, probably by decreasing the T lymphocyte that drives atopy. A recent double-blind, placebo-
controlled study demonstrated a statistically significant response in those on active therapy. This response was still present 3 years after discontinuing allergy vaccine therapy. Is allergy vaccine therapy the treatment of first choice? No?

If patients have rhinitis well controlled by avoidance, no other therapy is necessary. Nasal steroids would be the preferred mode of therapy for patients with nasal congestion and decreased quality of life. In those patients who have significant nasal and ocular symptoms, antihistamines may be preferred. Using both nasal steroids and antihistamines together adds little. When antihistamines or INCS fail, a trial of allergy vaccine therapy or a leukotriene receptor antagonist (LTRA) may be indicated.

Initial studies suggest thatLTRAs may be effective in rhinitis. The effectiveness may approach or equal that of some antihistamines. Some time will lapse before LTRAs are approved for rhinitis; however, most expect an approval by the Food and Drug Administration for LTRA in rhinitis in the next couple of years.

I hope the two articles appearing in this supplement to the JAOA are helpful to you in your practice. Both explore allergic rhinitis in depth. You may contact me if questions arise.

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References

Allergic rhinitis is estimated to affect as many as 40 million people in the United States on a regular basis, and even more individuals who have occasional symptoms. The disease is associated with a considerable burden on the healthcare system, accounting for a total of $7.9 billion in direct and indirect costs in 1997, and with significant adverse effects on patients’ quality of life, including disturbed sleep and impaired function at work and school. The pathophysiology of allergic rhinitis is complex, involving inflammatory mediators and immune cells that produce allergy symptoms via multiple mechanisms. The first principle of clinical management of patients with allergic rhinitis is avoidance of exposure to allergens, but this measure can be very difficult, and most patients require pharmacotherapy. Allergy vaccine therapy may be an appropriate and necessary option in selected patients with allergies refractory to other treatment modalities.

(Key words: allergen, allergy vaccine therapy, allergic rhinitis, allergy, avoidance, early-phase reaction, immunoglobulin E [IgE], immunotherapy, inflammation, late-phase reaction, mucosa, patient education, prevention, productivity, quality of life, rhinitis, sensitization, triggers)

Allergic rhinitis is the most common allergic disease in the United States, affecting 20 to 40 million people. It is an inflammatory disease of the upper airways, mediated by binding of antibodies to specific immunoglobulin E (IgE) antibodies, resulting in inflammation of the airway mucosa. People with such allergies have a large number of IgE antibodies that bind to specific antigens. They are said to be sensitized to those antigens, and in such patients, antigens that bind to IgE and cause allergic reactions are called allergens.

Allergic rhinitis is one category of rhinitis, the other being nonallergic rhinitis (Figure 1). Allergic rhinitis may be seasonal or perennial, but some patients may have both types. Typical seasonal allergens include grass, tree, and weed pollens.

Figure 1. Classification of rhinitis and symptoms and signs of allergic rhinitis. (Source: American Academy of Allergy, Asthma & Immunology. The Allergy Report. Available at: http://www.theallergyreport.org/reportindex.html. Accessed April 2, 2002.)