Allergic Rhinitis

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KEYWORDS

• Allergic rhinitis • Type 1 hypersensitivity • Vasomotor rhinitis • Skin prick testing
• Serum-specific Ig E • Allergen immunotherapy • Chronic rhinosinusitis
• Nasal polyps

KEY POINTS

- Allergic rhinitis affects up to 1 in 6 individuals, with health care costs estimated in the billions of dollars.
- Allergic rhinitis is a type 1 IgE-mediated hypersensitivity reaction and usually presents with typical symptomatology.
- Allergic rhinitis can be diagnosed by history and physical examination, with testing reserved for treatment of nonresponders or when identification of the specific cause is necessary to guide treatment.
- There are many therapeutic options, with intranasal corticosteroids the single most effective agent.

INTRODUCTION

Rhinitis is defined as inflammation of the nasal mucosa and affects up to 40% of the population. Among all causes of mucosal inflammation, allergic rhinitis (AR) is the most common, affecting 1 in 6 individuals. Symptoms of AR interfere with all facets of daily life and are associated with reduced quality of sleep and work performance.[1] Previously thought of as a disease restricted to the nasal passages, AR is now viewed as a manifestation of systemic airway disease and is often comorbid in patients with asthma. As a type 1 IgE-mediated hypersensitivity process, symptoms of AR are triggered by allergens; thus, minimizing allergen exposure should be an essential component of any treatment plan. AR often goes undetected by clinicians due to its nature as a long-standing condition. Patients suffering from AR may not seek medical treatment because they often fail to recognize its impact on their daily lives. As such, physicians should routinely screen for this widespread and debilitating condition with a focused history and physical examination.

EPIDEMIOLOGY

Approximately 10% to 20% of the global population suffers from AR, the most common cause of reversible nasal congestion. The reported prevalence of AR has

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Prim Care Clin Office Pract 43 (2016) 465–475
http://dx.doi.org/10.1016/j.pop.2016.04.009 **primarycare.theclinics.com**
0095-4543/16/$ – see front matter

been steadily increasing. The true incidence likely remains underestimated, however, because data collection hinges on physician diagnosis and misses those who are undiagnosed or self-medicate. In the United States, AR is the most common atopic condition, affecting between 9% and 16% of the population. Of patients with AR, 80% develop symptoms before the age of 20.[2] Further highlighting the significance of AR in the pediatric population, in the United States from 1994 to 2002, the prevalence of AR in more than 2000 children ages 13 to 14 years increased from 13% to 19%.[3]

The direct health-related cost expenditure of AR is estimated to be between $2 billion and $5 billion per year with an additional $2 billion to $4 billion lost in annual productivity.[4] A 2007 cohort of more than 8000 US workers revealed that AR caused greater loss of productivity than any other illness, including hypertension, diabetes, and heart disease, and accounted for approximately one-quarter of all lost productivity.[5] In 2006, AR accounted for more than 12 million office visits in the United States, making it the 16th most common primary diagnosis for outpatient office visits.[4] The prevalence and burden of AR obligates primary care providers to be able to readily and cost-effectively diagnose and manage this chronic condition.

CLINICAL PRESENTATION

The clinical presentation of AR is a consequence of its pathophysiology as a classic allergen response. Inflammatory mediators, including mast cells, macrophages, eosinophils, and lymphocytes, enter the nasal mucosa after introduction of the inciting allergen. The most common allergens include dust mite fecal particles, animal dander, molds, and pollens.[6] Identification of the inciting agent is not always necessary to effectively treat AR.

The structure and histology of the nose is designed to allow it to function as a wet filter. Its main purpose is to humidify inhaled air. To accomplish this, the nose must maintain an extensive vascular network allowing it to produce copious amount of mucous, which in the average adult nose averages 2 cups of mucous daily.[7] The mucous captures inhaled particles, gases, and vapors at which point ciliated cells direct them to the back of the throat, allowing them to be swallowed thus diverting them from the lower respiratory tract.[8] This postnasal drip is actually a normal physiologic process. Its consequences, however, such as pharyngitis, vocal cord dysfunction, and cough, indicate potential pathology.[7] When the captured particles incite the IgE-mediated allergy cascade, symptoms soon follow.

As a mucosal antibody, IgE is found in the lining of the eyes, nose, and lower airways. Basophils and mast cells are activated when an allergen bridges 2 specific IgE molecules. The principal vasoactive elements, including histamine, leukotrienes, and prostaglandins, are responsible for the initial phase of symptoms. Inflammatory cells, such as eosinophils, macrophages, and neutrophils, are then recruited, and their arrival leads to further release of vasoactive mediators, producing a delayed-phase inflammatory response. This second wave of symptoms occurs 4 to 6 hours after exposure to the inciting allergen.[9]

The classic symptoms of AR are nasal congestion, nasal itching, sneezing, and rhinorrhea. Allergic conjunctivitis presents as itchy, watery eyes resulting from the same pathophysiology as AR and is not surprisingly a common comorbid condition. Patients may not attribute their symptoms to seasonality or surrounding environments and present with what they believe to be a viral-related illness. Patients' degrees of atopy vary from minimal to severe and symptoms of AR exhibit a similar pattern. For many patients, AR-induced nasal congestion is not merely a trivial stuffy nose but a debilitating problem. A 2007 prospective, cross-sectional survey, identifying