Allergic Rhinitis: Nothing to Sneeze At

Elizabeth F. Jaffe, M.D., Ph.D.

Timber Lane Allergy & Asthma Associates
South Burlington, VT
Clinical Associate Professor, Department of Pediatrics, Fletcher Allen Health Care
About Dr. Jaffe

Elizabeth Jaffe, M.D., Ph.D., is a clinical associate professor of pediatrics at the University of Vermont. Dr. Jaffe earned her medical degree and her Ph.D. in genetics at the University of Washington in Seattle. She completed a residency in pediatrics at the University of Vermont and a fellowship in allergy and immunology at McGill University in Montreal. Dr. Jaffe joined the UVM/FAHC faculty in 2007, and is a board-certified allergist in clinical practice at Timber Lane Allergy and Asthma Associates.
Allergic Rhinitis - Definition

- Inflammation of membranes lining the nose
- Inappropriate inflammatory response to common environmental allergens
- Triggered by an antibody, IgE, specific for the allergen
Symptoms

• Early and late phase responses
• Runny nose, sneezing, nasal and ocular itchiness, congestion seen with “acute” exposure
• Nasal congestion more prominent with prolonged exposure
ARIA Classification

- Intermittent - symptoms present <4 d/wk or <4 consecutive weeks
- Persistent > 4d/wk or >4 consecutive weeks
- Mild - none of the following
  • Sleep disturbance
  • Impairment school or work
  • Impairment daily activities, leisure, sport
  • Sx present but not troublesome
- Mod/severe 1 or more of above
Prevalence

- Affects up to 10-30% of adults in U.S.

- Up to 40% of children
  - (AAAAI, Allergy Report Overview of Allergic Disease, 2000)
  - If both parents atopic, risk 50%

- Prevalence rates have been rising over past 2-3 decades
Sanitation Hypothesis

- **B cell**
- **IgE**
- **bacteria, viruses, immunotherapy**

**TH0**
- **TH1**
  - IFN-gamma, IL-12
  - **Macrophage**
  - Ingest bacteria, infected cells

**TH2**
- **HISTAMINE**
- **IL-4, IL-13**
- **B cell**
- **IgE**

**atopy**
IgE-Dependent release of inflammatory mediators. IgE binds to high- and low-affinity receptors (FcRI or FcRII) on effector cells. The inflammatory cascade is initiated when a critical mass of IgE antibodies bound to effector cells is cross-linked by allergen. This results in the degranulation of effector cells and the release of a comprehensive array of mediators that are causally linked to the pathophysiology of allergic disease.
Allergic Rhinitis Impact

• One of the most common chronic diseases

• Importance often underplayed

• Often self-treated by patients with medications as debilitating as symptoms
Impact of Allergic Rhinitis

- Significant impairment at work
  - 3.4 million missed work days per year in US

- School
  - 2 million missed school days in US per year because of sx of AR (EO Meltzer, J Occup Med 1990;32:327-34)

- Decreased quality of life
Allergies in America Survey

- 2500 phone interviews age >18 diagnosed with allergic rhinitis (2006)
- 500 children with allergic rhinitis (2007)

• 1/3 children and 2/5 adults intolerable discomfort without treatment
• 50% dissatisfied with their current treatment
• Nasal congestion most bothersome symptom
• Impact on quality of life similar to asthma

Burden of Allergic Rhinitis, Allergy and Asthma Proceedings, 2007, V. 28
Why is AR hard to control

- Many different inflammatory molecules
- One medication often does not suffice
- Can have “priming”. Allergy to spring pollen can make symptoms worse with later pollen seasons
Important Comorbidities

- Otitis Media with Effusion
- Upper Respiratory Infection
- Nasal Polyposis
- Allergic Rhinitis
- Asthma
- Sinusitis
Linked airway

• Allergic rhinitis (AR) and asthma often coexist
  – Inflammation often occurs throughout the airway, yet if one disorder predominates, the other often goes unrecognized
  – Cough in person with runny nose not always post-nasal drip
Linked Airway

Central Sensitization and Nasopharyngobronchial Reflexes

- Drainage of Inflammatory Material
- Systemic Propagation of Nasal Inflammation

- Air Warming and Humidification
- Particle / Irritant Trapping
- Nitric Oxide
Triggers of Allergic Rhinitis

• Seasonal Allergic Rhinitis
  – Pollens and molds
  – Accounts for approximately 20-30% AR
  – Pollen allergy need at least 2 seasons to develop

• Perennial Allergic Rhinitis
  – Dust mite, animal danders, cockroach
  – Can develop in first year of life
Dust Mites

• Features
  • Human host
  • Feces source of allergens
  • Sensitization associated with increased asthma risk

• Environmental control
  • Impermeable encasings for pillow, mattress
    – Mite colonization within weeks with new mattress or pillow
  • Reduce upholstered furniture
  • Low humidity (die at<50% humidity)
  • Bedding, stuffed animals wash in hot water
  • Remove carpets
Animal Danders

- Cats/Dogs (60% of US homes have pet)
  - No substitute for removing pet from home
  - Cat and dog allergens can be transferred on clothes and in hair
  - Wash animal **twice a week** significantly reduces dander
  - Higher levels in carpeted areas

- Rodents extremely allergenic
  - Urine main source of allergen/have persistent proteinuria
  - Guinea pigs, hamsters, rabbits not appropriate classroom pets
What REALLY happens when you leave for work!
Pollens/Molds

• Seasonal symptoms
  – In Vermont:
    • Tree pollen (mid April-early June)
    • Grass pollen (May-July)
    • Ragweed (August to frost)
    • Alternaria mold (peaks with ragweed)

• Oral Allergy Syndrome also clue to pollen allergy

• Avoidance
  – DON’T MOW THE LAWN!!!
  – AC preferable to open windows and fans
Birch Pollen
Birch pollen micro
Grass pollen
Grass pollen micro
Ragweed
Ragweed pollen micro
Physical exam

- Pale swollen nasal mucosa
- Mouth breathing
- Nasal crease (allergic salute)
- Infraorbital crease
- Allergic shiner (least specific)
- Injected conjunctivae
- Chemosis
Chemosis
Laboratory Diagnosis

• **Blood tests**
  – Total IgE not very helpful
  – IgE for specific allergens (RAST test)
    • Less specific than skin tests, more expensive
    • Use if skin testing unavailable, can’t get off antihistamines, or widespread eczema

• **Skin test**
  – Sensitive/specific with good quality extracts, flexible
Why Allergy Test

• Confirm allergy
  – 50% rhinitis not allergic
  – Large differential including adenoid enlargement, vasomotor rhinitis, gustatory rhinitis, nasal polyps, drug-induced (OCP, ACE-I, ASA), occupational

• Elicit sensitivity not detected by history

• Demonstrate effect of specific allergen to patient and/or family
Major Treatment Options

- Environmental control “More is less”
- Nasal corticosteroids-use for nasal congestion, sneezing, rhinitis, pruritis
- Antihistamines-use for sneezing, rhinitis, pruritis
- Leukotriene antagonists
- Immunotherapy
- Nasal cromolyn
  - 4-7 d to start working, full benefit takes weeks
- Oral and nasal decongestants
  - Insomnia, irritability, heart palpations, hypertension, addiction to nasal decongestants
Nasal Steroid Sprays

• First-line therapy for allergic rhinitis

• Effective for nasal congestion, in addition to runny nose, sneezing, pruritis

• More effective than the combination of an antihistamine and leuktriene antagonist
Nasal Steroids (cont)

• Generally 12 hours until onset, may start as early as 3-4 hours in some patients
  – Fluticasone (Flonase, Veramyst)
  – Mometasone (Nasonex)
  – Budesonide (Rhinocort)
  – Triamcinolone (Nasacort)
  – Ciclesonide (Omnaris)

• Best used daily, but can be effective even if used as needed
• Helps eyes as much as oral antihistamine
• Can help for nonallergic rhinitis
• Nasal irritation minimized by aiming towards ears
• Safe
Treatment-H1-antagonists

– First Generation:
  • Diphenhydramine (Benadryl) introduced in 1946; was 1st-line tx for AR for decades
  • Can give sedation, stimulation, dry mouth
  • Performance impairment demonstrated even in individuals who do not feel impaired

– Second or Third Generation Preferred
H1-antagonists (cont)

• Non-sedating oral Antihistamines
  • Cetirizine (Zyrtec)
  • Levocetirizine (Xyzal)
  • Fexofenadine (Allegra)
  • Loratadine (Claritin)
  • Desloratadine (Clarinex)

• Nasal Antihistamines
  • Azelastine (Astelin)
  • Olopatadine (Patanase)
  – Generally well-tolerated; do not have cardiac side effects
  – Once to twice daily therapy vs. 4-6 hours for 1st generation
  – Not help nasal congestion as well as nasal steroids
  – Not help non-allergic rhinitis
Leukotriene Antagonists

• Montelukast (Singulair)
• Can help with nasal congestion, runny nose, but generally not as effective as nasal steroid sprays
• Consider in patients with both asthma and allergic rhinitis
Immunotherapy (Allergy Shots):

• Not a Bandaid

• Has a unique position in chronic disease

• The only treatment capable of long-term change to the immune response
  – Reduces inflammatory molecules
  – Makes blocking antibodies that block IgE binding
  – Increases Suppressor T cells
IT Technique

• Subcutaneous injections

• Start at 1/100,000 to 1/10,000 of eventual maintenance dose

• Building phase 6 mo with weekly shots, then 3-5 years of monthly shots
IT Indications

- Well-established treatment for allergic rhinitis and conjunctivitis
- Standard of care for treatment of venom systemic reactions
  - Reduces chance of systemic reaction on subsequent sting from 60% if untreated, to less than 2% during maintenance treatment
- Can also decrease asthma symptoms
Immunotherapy as Preventative Treatment for Asthma
Moller C et al. JACI 2002; 109:251-256

- Children, ages 6-14, with symptomatic grass and/or birch pollen allergy
- Received either 3 years of specific immunotherapy or no injections
- 151 children without asthma at start of study
- Found significantly higher rate of asthma development in those who did not have allergy shots
IT May Protect Against Asthma Development

![Bar chart showing the percentage of patients with and without asthma in SIT and control groups.](image)

- **SIT**: N=60
  - No asthma: N=19
  - Asthma: N=40

- **Control**: N=32
  - No asthma: N=19
  - Asthma: N=40

Odds-ratio = 2.52
Early Immunotherapy May Prevent New Sensitizations

Des Roches A et al. JACI 1997;99:450-453

- 44 children, ages 2-6, only allergic to dust mite
- Half received IT with dust mite for three years, half only medications and environmental control
- 10/22 of IT-treated group had no new allergies after three years
- All of untreated group had new allergies (other positive skin tests) after three years
Immunotherapy-Risk/Benefit:

• Benefits:
  – Relief of symptoms, decrease in medication
  – May be effective in some for decades
  – May prevent development or progression of asthma and allergic rhinitis

• Risks
  – Systemic reactions
    • Minimize by slow build-up phase
    • Not administering when asthma is unstable, or severe AR symptoms
Immunotherapy Summary

- Effective control of atopic disease requires a global approach
  - Currently IT is used for refractory disease

- Preventative immunotherapy is not yet recommended in this country, but may be a future first-line treatment as safer immunotherapy is developed
Allergic Rhinitis-Summary

• One of the most prevalent chronic diseases
• Importance underplayed
• Has important associated diseases
• Can use stepwise tx:
  – Environmental control essential
  – Nonsedating AH and/or nasal steroid
  – Immunotherapy if above measures do not control symptoms
  – Steroid pulse in season if severe symptoms