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CLINICAL PEARLS UPDATE#24

Rhinitis and Sinusitis

March 8, 2004

Dear Colleague,

Our clinical pearl this week is on the role of immune burdens in **rhinitis and sinusitis**. Both conditions reflect cumulative repair deficits (clinically known as inflammation) and increased host hospitality to infection and/or delayed allergens. Now, with the benefit of **LRA by ELISA/ACT® assays and treatment guide**, you have available a **patient-specific first line, comprehensive care approach to chronic rhinitis and sinusitis**. This includes the environmental chemicals, molds, and aspirin hypersensitivities that can be involved.

We encourage you to share this valuable clinical update newsletter with your colleagues and staff so they can learn more about how our comprehensive approach can be applied to their practice with beneficial results. Please also let us know if any of your colleagues or staff would like to be added to our email distribution list.

We are grateful for the opportunities to be of service to you and your patients.

Sincerely,

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Lab Director

Borish L. **Allergic rhinitis: systemic inflammation and implications for management.** *J Allergy Clin Immunol* 2003;112(6):1021-1031.

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Allergic rhinitis triggers a systemic increase of inflammation. Within minutes of allergen exposure, immune cells release histamine, proteases, cysteinyl leukotrienes, prostaglandins, and cytokines. Some produce the early symptoms, while others augment the production, systemic circulation, and subsequent infiltration of the nasal mucosa with inflammatory cells that sustain the symptoms. Systemic circulation of inflammatory cells permits their infiltration into other tissues where chemoattractant and adhesion molecules already exist. Consequently, allergic rhinitis is linked to comorbid conditions: asthma, chronic hyperplastic eosinophilic sinusitis, nasal polyposis, and serous otitis media. Effective therapy should be directed at underlying inflammation and its systemic manifestations. It should improve the rhinitis and the comorbid conditions. Antihistamines relieve early symptoms by blocking basophil- and mast cell-generated histamine, but they do not significantly influence the pro-inflammatory loop. They are often little better than placebo. Oral corticosteroids provide the systemic anti-inflammatory efficacy, but their toxicity precludes such an approach. Intranasal corticosteroids effectively target the local inflammatory processes of rhinitis, reducing local inflammatory cells within the nares, but they do not directly access tissues involved in the comorbid conditions. Leukotriene modifiers have both systemic anti-inflammatory effects and an acceptable safety profile.

Slavin RG. **Resistant rhinosinusitis: what to do when usual measures fail.** *Allergy Asthma Proc* 2003;24(5):303-306.

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There are some patients with rhinosinusitis in whom the condition never clears or it recurs after a short period of time. The clinician must consider underlying conditions that must be brought under control to arrive at a satisfactory outcome. These conditions include allergy, immune deficiency, cystic fibrosis, gastroesophageal reflux, and structural abnormalities. Other disease states are important to consider because they may mimic bacterial rhinosinusitis but require other therapeutic approaches. These include chronic hyperplastic eosinophilic rhinosinusitis, fungal sinusitis, and aspirin sensitivity.