Sinusitis and Asthma
Associations, Influences, and Principles of Management

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CONTENTS

POSTULATED MECHANISMS
EXPLAINING THE RELATIONSHIP
BETWEEN SINUSITIS AND ASTHMA
THE CLINICAL ASSOCIATION BETWEEN SINUSITIS AND ASTHMA
MICROBIOLOGY OF THE UPPER AND LOWER RESPIRATORY TRACT
PRINCIPLES OF DIAGNOSIS AND MANAGEMENT
UNIQUE ASSOCIATION OF SINUSITIS AND ASTHMA
SUMMARY
REFERENCES

Chronic nocturnal cough, or worsening of asthma may be associated with sinusitis. Until proven otherwise, the treatment of sinusitis should be considered as first-line intervention.

A physiologic unity between the upper and lower airway of humans has been recognized throughout the 20th century. A number of studies have addressed and described a relationship between the inflammatory diseases occurring in the upper respiratory tract, especially sinusitis, and its consequences in the thoracic airways. At the beginning of this century, Bullen (1), Gottlieb (2), and Weille (3) found coexistent sinusitis in 20–70% of a large number of adult asthmatics. After reviewing 1074 cases of asthma, Rackemann and Tobey in 1929 wrote that “lesions in the nose and sinuses...may develop from the same fundamental cause as the asthma itself” (4). More recent studies have found that 50–70% of children and adults with asthma have radiographic evidence of sinusitis (5,6).

Schwartz et al. examined the sinus radiographs of 217 patients with a flare-up of asthma symptoms and found abnormal sinus radiographs in 47%, as compared with 29% of patients with rhinitis as their sole complaints (7). Fuller et al. have indicated that 75% of patients admitted to Los Angeles Children’s Hospital for status asthmatics had abnormal sinus radiographs (8). The majority of evidence revolves around the impressive coincidence of these two entities, as well as the observations that asthma improves, sometimes dramatically, after medical or surgical management of coexisting sinusitis (9–11). Despite these observations, there remains limited conclusive information whether upper respiratory tract inflammation and sinusitis specifically contribute to the pathogenesis of asthma, or whether the two problems simply “coexist” because of a common pathogenesis.

 Interruption of the normal functioning of the upper airway has been shown to influence reactivity of the lower airway. The nose is our most sophisticated “air conditioner.” It filters the inspired air and captures large particles by the hairs within the nostrils, whereas other noxious
substances are trapped in the mucus. If nasal obstruction occurs, an increased burden of allergens and/or irritants will be delivered to the lower airways with hyperresponsiveness as a potential outcome. The nose heats and humidifies the air through the highly vascularized mucosa of the turbinates and septum. If the nose is blocked, cooler, drier air will be delivered to the lungs, which can trigger or potentiate exercise-induced asthma in the susceptible host.

Furthermore, it has been shown that chemical or mechanical irritation of the nose can induce bronchoconstriction. Exactly how and to what extent such an excitatory system occurs is unknown, and a discussion of such postulates is beyond the scope of this chapter. This phenomenon appears to be more severe in patients with coexisting allergic rhinitis and asthma (12). Kaufman et al. have demonstrated a beneficial effect from trigeminal resection when reflex bronchospasm is induced by nasal and nasopharyngeal irritation (13). Observations such as these appear to confirm Sluder’s original hypothesis in 1919 of the existence of a nasal-bronchial reflex in humans (14). The theory of a nasobronchial reflex contends that reflex bronchospasm occurs via stimulation of neural fibers in the nasal mucosa, which then triggers a trigeminal-afferent-vagal efferent neural arc (15).

Considerable evidence has been published demonstrating an increase in lower airway responsiveness in nasal hypersensitivity diseases. Using a ragweed bronchial inhalation challenge model, there exists considerable overlap in lower airway hyperresponsiveness between ragweed allergic patients with asthma and those with allergic rhinitis alone (16). Using increased sensitivity to methacholine or hyperventilation as a measure of more nonspecific bronchial hyperresponsiveness, a significant percentage of patients with rhinitis alone can be shown to be reactive in a range typically seen among asthmatics (17). Individuals with nasal polyps will have an estimated 25–30% risk of ultimately developing asthma (18). Attempts at demonstrating bronchial hyperreactivity with methacholine challenge in nasal polyposis have, however, been inconclusive (19,20). Nevertheless, the incidence of asthma appears to increase steadily as one progresses from nasal polyposis to associated rhinosinusitis to a history of acetylsalicylic acid (ASA) intolerance (ASA hypersensitivity tetrad). Although none of these studies illustrate a common mechanism whereby diseases of the nose and sinus will induce asthma, they do emphasize the need to look at the upper and lower airway as systems capable of being influenced by one another.

**POSTULATED MECHANISMS EXPLAINING THE RELATIONSHIP BETWEEN SINUSITIS AND ASTHMA**

In 1925, Gottlieb postulated four possible mechanisms whereby inflammation of the sinuses could aggravate bronchospasm (2):

1. Mucopurulent postnasal drainage leading to continuous infection of the trachea and bronchi;
2. Absorption of toxic products from retained purulent material in the sinuses triggering an immunologically mediated asthmatic reaction;
3. Sinusitis inducing nasal obstruction, which, in turn, leads to mouth breathing of cold, dry air inducing bronchospasm; and
4. Nerve reflex bronchospasm via irritation of the nasal ganglion.

Despite our improved knowledge of the physiology and immunology of the naso-sino-bronchial system, these concepts, born almost three-quarters of a century ago, remain the fundamental postulates upon which our understanding of the relationship between sinusitis and asthma stands.

**Aspiration of Infected Material**

Bardin and colleagues attempted to mimic the aspiration postulate in asthmatics with sinusitis (21). They instilled a radionuclide in the maxillary sinus of four patients with maxillary sinusitis, and nine patients with sinusitis and asthma during needle puncture of the sinus. In all patients, the radionuclide could be demonstrated in the nasopharynx, sinus, and gastrointestinal tract over the next 24 h, but not in the lower airways. This negative study suggests that aspiration is not a factor in the coexistence of asthma and sinusitis.

**Eosinophils**

Eosinophils have unique biologic features that allow them to be major contributors to the inflammatory process: the preformed granule-associated proteins, including eosinophil peroxidase, major basic protein, cationic protein, and eosinophil-derived neurotoxin, have been identified. Major basic protein is most relevant and is capable of...