3 Clinical and Allergic Evaluation of the Patient with Bronchial Asthma

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Key Points

• A diagnosis is usually in the details.
• Asthma is primarily an inflammatory disease of the bronchi with a bronchospastic component.
• The symptoms of bronchial asthma will suggest a wide variety of clinical conditions; the history is critical in defining etiology.
• The history should focus on seasonality, associated factors, current medications and other illnesses under treatment.
• Anosmia (loss of sense of smell) and/or hyposmia (a reduction in an ability to smell) are frequently symptoms of sinusitis with consequent asthma.
• Aspirin/Nsaid sensitivity suggests triad asthma, i.e., nasal polyps/asthma/Nsaid sensitivity.

From: Bronchial Asthma: Principles of Diagnosis and Treatment, 4th ed.
M. E. Gershwin and T. E. Albertson, eds. © Humana Press, Totowa, NJ
• Physical exam focuses on the nasal airway, i.e., polyps, turbinate swelling, septal perforation, and the chest, i.e., wheezing and degree of expiratory obstruction.
• Even in patients with a clear chest, a spirometric study is critical in assessing acute/chronic respiratory complaints to define the degree of obstruction.
• Sinus radiographs, i.e., sinus CT and x-rays, are underutilized in defining sinusitis as an etiology for acute and chronic asthma.
• An assessment of IgE mediated sensitivity, i.e., allergies, should be conducted in any asthmatic with a seasonal/exposure related history not only to confirm the diagnosis but also to initiate appropriate environmental control.
• Food sensitivity is rarely a cause of bronchial asthma.
• Recurrent cough/wheezing in an older/obese patient suggests G-E reflux even in the absence of upper GI complaints.

Introduction

Few diseases have come under more scrutiny and redefinition as bronchial asthma. Originally conceived as a bronchospastic condition with psychiatric overtones, and treated primarily with β-agonists/xanthines/sedatives, bronchial asthma is now considered an inflammatory disease of the airways, with bronchospasm, i.e., reversibility, representing a secondary but identifying feature. This is not to suggest that inflammation had not been identified as an associated feature. Unfortunately, the disease came under the microscope only during autopsies, primarily in patients who had died of status asthmaticus. The inflammation defined both on gross and microscopic examination was felt to represent a terminal event. It is tempting to postulate that the genesis of the data, which holds that the inflammatory component represents a complex interaction between mediators, neurons, effector cells, and triggering proteins/haptens, may have been spurred by the response of the disease, 45 yr ago, to the newly discovered corticosteroids. Nonetheless, despite the obvious saluatory effect of an oral/inhaled anti-inflammatory agent it was difficult to envision that nonspecific inflammation, a response long deemed beneficial and healthful, especially in responding to bacterial/viral invasion, could be responsible not only for asthma, but for a host of other inflammatory conditions, now termed autoimmune/connective-tissue diseases.

Research has now gone beyond the histopathology of the inflammatory response. Researchers find themselves in a molecular labyrinth of cytokines, interleukins/chemokines, sophisticated physiology, and a hereditary predisposition, all of which are detailed in subsequent chapters. These findings have, furthermore, led not only to an overall dramatic change in the pharmacotherapy of the disorder, but to the development of specific drugs that go to the molecular basis of inflammation.

The diagnosis of asthma in any given patient occurs after a thorough historical assessment of the patient’s complaints, directed physical examination, specific evaluative studies, and, possibly, their response to a variety of therapies. The patient in whom the diagnosis is confirmed will characteristically report a plethora of