



A CLOSER LOOK AT ASTHMA

by *Bill Wojciechowski, MS, RRT*

Asthma can be defined as a chronic inflammatory disease of the tracheobronchial tree, characterized by airway hyperresponsiveness to a variety of antigens or stimuli. These stimuli cause bronchoconstriction, chest tightness, wheezing, increased mucus production and air flow limitation, all of which are reversible spontaneously or through pharmacologic intervention.

The inflammatory component of asthma has taken center stage in recent years. This pathophysiologic process appears to be responsible for the adverse effects associated with asthma. Not too long ago, bronchoconstriction was the primary focus of asthma control. Even though bronchoconstriction remains a target of asthma control, airway inflammation is now considered the major pathophysiologic villain.

This disease can have a devastating impact on the quality of life of those stricken with it, and the economic impact of asthma on society is enormous. Recent pharmacologic advancements have provided benefits in the treatment and control of asthma. Nonetheless, the prevalence of this disease has risen.

Epidemiology

Asthma is a worldwide disease. It is ubiquitous in its occurrence. In the U.S. alone, asthma afflicts more than 30 million people, including 6 million children. Data have shown that, since 1991, deaths related to asthma have declined despite the increased prevalence of the disease.

According to the American Academy of Allergy, Asthma, and Immunology, about 5,000 asthma-related deaths are attributed to asthma each year. Furthermore, in the face of increased asthma incidence, fewer patients with asthma claim to have physical restrictions from the disease. The past 10 years have shown a stabilization of hospitalization rates; however, children between the ages of 0 to 4 years have experienced an increased rate.

Ethnic and racial disparities in asthma persist. According to the National Center for Health Statistics (NCHS), Puerto Ricans have the highest rate of lifetime asthma (196 per 1,000) and Mexicans the lowest (61 per 1,000). A survey conducted by the NCHS revealed that Puerto Ricans were almost 80 percent more likely to have ever been diagnosed with asthma compared to non-Hispanic whites. Non-Hispanic blacks and American Indians were about 25 percent more likely to have ever been diagnosed with asthma compared to non-Hispanic whites.

Females were about 7 percent more likely than males to have ever been diagnosed with asthma. Among children ages 0 - 17, however, males were more likely to have an asthma diagnosis.

All asthma patients have chronic airway inflammation in common. Inflammation is a normal physiologic response to injury or infection, and it is considered to be a non-specific immune response. The inflammatory response mobilizes components of the immune system — neutrophils, eosinophils, chemotactic peptides and mononuclear cells — and summons them to the site of injury or infection.

What also occurs is increased blood delivery (hyperemia) to the site of injury, as well as vascular permeability in that region. Essentially, immune system components leave the vasculature. The white blood cells involved in this non-specific immune response perform phagocytosis (ingestion of debris and infectious agents), and in the process produce oxidative bursts and degranulation of lysosomal contents. Both of these contribute further to the development of inflammation.

Mast cells in the lungs play a prominent role in the inflammatory process. They are situated in various locations in the lungs, including beneath the basement membrane, in proximity to blood vessels in the submucosa, adjacent to submucosal glands, scattered throughout bronchial smooth muscle, in the interalveolar septa and in the bronchial lumen. They are constituents of the body's immune system. Because of their location, mast cells are in a position to react immediately to allergens and various stimuli.

Mast cells have receptors throughout their surface. These receptors are specific for antigens to which a person is hypersensitive. When a person comes in frequent contact with a certain antigen or antigens to which the body has formed immunoglobulin (IgE) antibodies, antigen-antibody reactions occur along the surface of mast cells, causing the cells to degranulate. Mast cell degranulation causes the release of many potent mediators.

Some of these mediators are preformed and others (eicosanoids) are formed at the time of degranulation. Some of the

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mediators are histamine, heparin, leukotrienes, serotonin, chemotactic factors, tryptase, chymase and prostaglandins. Mast-cell activation also stimulates the arrival of other inflammatory cells. Subsequently, in addition to further inflammation, mast cell degranulation causes bronchial smooth muscle contraction, increased mucus production, all of which obstruct the flow of air throughout the lungs.

Ordinarily, inflammation represents a part of the healing process whenever the body responds to injury or infection. The disappearance of inflammation signifies that healing is complete. However, in the case of asthma, inflammation does not completely resolve. The result is recurring attacks.

Uncontrolled inflammation associated with asthma is believed to result in airway remodeling. Airway remodeling involves permanent alterations of the architecture of the airways. These anatomic alterations include hypertrophy and hyperplasia of bronchial smooth muscle, mucous gland hypertrophy, hypersecretion of mucus, deposition of connective tissue in the airway walls and thickening of the basement membrane. All of these structural changes increase the thickness of the airway walls and narrow the lumen of the airways further. The ultimate consequence is significantly impaired airflow that worsens during exacerbations.

The classic signs and symptoms of asthma are cough, shortness of breath, wheezing, chest tightness and mucus production. These signs and symptoms range from mild to severe. They may also occur intermittently or continuously.

Some people with asthma may have mild symptoms such as occasional wheezing and experience sporadic exacerbations. Between exacerbations, these patients often breathe with no difficulty and have no physical limitations. Others experience coughing and wheezing throughout the day, as well as at night. They tend to have severe dyspnea, and are essentially incapacitated.

The signs and symptoms alone do not provide enough information to diagnose asthma. What confounds the diagnosis of asthma is that patients frequently are asymptomatic when they visit the physician. When a person exhibits the signs and symptoms, suspicions are raised, and a thorough medical history and physical exam are conducted. Any relationship between signs and symptoms and patient allergies should be explored. In many instances, even when signs and symptoms, medical history and physical exam are indicative of asthma, confirmation is generally withheld until pre- and post-bronchodilator studies are performed. If the person demonstrates an improvement in the FEV1 by at



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least 200 mL and 12 percent, the diagnosis of asthma is made. Spirometry may not always reflect the presence of airflow limitation.

In these cases, a bronchoprovocation test may be conducted. This test involves challenging the subject's airways with progressively increasing doses of inhaled methacholine. Spirometry is performed after the inhalation of each dose. A drop in the FEV1 by 20 percent compared to the patient's control FEV1 signifies the presence of asthma. On the other hand, if spirometry remains normal throughout the bronchoprovocation test, the person likely does not have asthma.

Allergy testing is sometimes performed to identify if a patient is allergic to a specific antigen or antigens. Allergens often trigger the signs and symptoms of asthma. Therefore, patients who have allergic asthma must be cognizant of these factors in their environment in order to learn to avoid them. Not only can these allergens elicit asthma signs and symptoms, they may also cause an exacerbation.

In the future, diagnosing asthma may be as simple as measuring the nitric oxide (NO) level in a person's exhaled breath. The fact that a patient with asthma has a greater concentration of exhaled NO than a normal person has been established. In fact, an asthma patient's exhaled NO concentration is roughly three times that of a normal person. Furthermore, studies have revealed that a patient with asthma having an asthma attack has an exhaled NO concentration twice that of a patient whose asthma is controlled.

Interestingly, other research has demonstrated that an asthmatic's exhaled NO concentration decreases when the person is treated with an inhaled corticosteroid. The speculation is that the amount of exhaled NO concentration is linked directly to the degree of airway inflammation.

In the next issue of Focus, "A Closer Look at Asthma, Part II" will cover different types of asthma and pharmacologic approaches to treating this disease.

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