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Extended Abstract

Allergic Rhinitis: Pearls of wisdom

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Abstract

This provides an overview of Allergic Rhinitis and its management. It is very useful for students of Rhinology and clinicians managing this disease. It introduces them to a systematic approach of assessing allergic rhinitis patients which is very commonly found in most populations and causes considerably morbidity. Allergy per se is a very difficult subject to master and it is with great perseverance one can treat patients suffering from this condition. The cornerstone of managing a patient of allergic rhinitis is first and foremost obtaining a good history. This is to be followed by a thorough examination and investigations. The general practitioner is the first expert to be involved in management of allergic rhinitis patient followed by specialists in particular otorhinolaryngologists, and finally by allied healthcare personnel. Inflammation of nose and paranasal sinuses is characterized by two or more symptoms- namely, nasal blockage / obstruction / congestion or nasal discharge. Associated symptoms include facial pain / pressure and either reduction or loss of smell. Certain diagnostic endoscopic signs of nasal polyps and or mucopurulent discharge and or mucosal oedema in the middle meatus and or CT changes of mucosa within the ostoemeatal complex, and or sinuses are seen. Definitions, aetiologies, clinical presentations, diagnosis / prognosis and management of allergic rhinitis is dealt with. Common allergens causing the disease are mentioned, pathophysiology and classification of allergic rhinitis is discussed in detail. Different types of allergen testing are highlighted along with their specific role and uniqueness. Principles of immunotherapy in treatment of allergic rhinitis are discussed here. Health effects of allergic rhinitis along with its impact on physical quality of life is mentioned. The basic idea of this presentation is to improve diagnostic accuracy by promoting appropriate use of ancillary tests like nasoendoscopy, allergy testing, computed tomography etc. and reduce inappropriate antibiotic use. The basic treatment plan of allergic rhinitis is according to the severity and duration. It consists of allergen avoidance, pharmacotherapy, allergen immunotherapy and surgery which has limited role.

Allergic rhinitis (AR) is an atopic disease characterized by symptoms of nasal congestion, clear rhinorrhea, sneezing, postnasal drip, and nasal pruritis. It affects one in six individuals and is associated with significant morbidity, loss of productivity, and health-care costs. Historically, AR was thought to be a disease process of the nasal airway alone. Still, the development of the unified airway theory has classified AR as a component of systemic allergic response, with other associated conditions, such as asthma and atopic dermatitis, sharing an underlying systemic pathology.[1] AR can be classified as either seasonal (intermittent) or perennial (chronic), with approximately 20% of cases being seasonal, 40% perennial, and 40% with features of both.[2] In addition to nasal symptoms, patients with AR may also present with associated allergic conjunctivitis, non-productive cough, Eustachian tube dysfunction, and chronic sinusitis. Once diagnosed, AR is treatable with a variety of modalities, with intra-nasal glucocorticoids being first-line therapy.

The allergic response is classified into early and late phase reactions. In the early phase, allergic rhinitis is an immunoglobulin (Ig)E-mediated response against inhaled allergens that cause inflammation driven by type 2 helper (Th2) cells.[2] The initial response occurs within five to 15 minutes of exposure to an antigen, resulting in degranulation of host mast cells. This releases a variety of pre-formed and newly synthesized mediators, including histamine, which is one of the primary mediators of allergic rhinitis. Histamine induces sneezing via the trigeminal nerve and also plays a role in rhinorrhea by stimulating mucous glands. Other immune mediators such as leukotrienes and prostaglandins are also implicated as they act on blood vessels to cause nasal congestion. Four to six hours after the initial response, an influx of cytokines, such as interleukins (IL)-4 and IL-13 from mast cells occurs, signifying the development of the late phase response. These cytokines, in turn, facilitate the infiltration of eosinophils, T-lymphocytes, and basophils into the nasal mucosa and produce nasal edema with resultant congestion.