ETHIOPIAN INTERNATIONAL JOURNAL OF MULTIDISCIPLINARY RESEARCH

eISSN: 2349-5715 pISSN: 2349-5707

Volume: 12, Issue 01, January-2025 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980 2024: 7,662

Jaxbarova X.J.

Andijan State Medical Institute,Assistant

ALLERGIC RHINITIS AND IMMUNE SYSTEM

Abstract. Allergic rhinitis (AR) represents a global health concern where it affects approximately 400 million people worldwide. The prevalence of AR has increased over the years along with increased urbanization and environmental pollutants thought to be some of the leading causes of the disease. Understanding the pathophysiology of AR is crucial in the development of novel therapies to treat this incurable disease that often comorbids with other airway diseases. Atopic allergic sensitization is defined by the production of immunoglobulin E (IgE)-mediated immune response toward allergens. Allergic rhinitis (AR) is a common disorder that afflicts 400 million people worldwide and it represents a global concern as its prevalence has increased over the years [1]. AR usually comorbids with other diseases such as asthma [2], leading to impaired quality of life, school or work performance, and significant financial impact. AR is shown to be caused by aberrantly high Th2 cytokines, and recent findings on the cause of AR are directed toward impairment of the nasal epithelial barrier integrity [3]

Keywords: allergic rhinitis, epidemiology, diagnostic criteria, pathophysiology, Th2 responses, immunity

Allergic rhinitis prevalence has increased significantly since the 1990s . It is reported to affect approximately 25 and 40% of children and adult globally, respectively. Approximately 80% of AR symptoms develop before the age of 20 years and peak at age 20–40 years before gradually declining. Allergic rhinitis is a very common disorder that affects people of all ages, peaking in the teenage years. It is frequently ignored, underdiagnosed, misdiagnosed, and mistreated, which not only is detrimental to health but also has societal costs. Although allergic rhinitis is not a serious illness, it is clinically relevant because it underlies many complications, is a major risk factor for poor asthma control, and affects quality of life and productivity at work or school[4] . Management of allergic rhinitis is best when directed by guidelines. A diagnostic trial of a pharmacotherapeutic agent could be started in people with clinically identified allergic rhinitis; however, to confirm the diagnosis, specific IgE reactivity needs to be recorded. Documented IgE reactivity has the added benefit of guiding implementation of environmental controls, which could substantially ameliorate symptoms of allergic rhinitis and might prevent development of asthma, especially in an occupational setting. Many classes of drug are available, effective, and safe. In meta-analyses, intranasal corticosteroids are superior to other treatments, have a good safety profile, and treat all symptoms of allergic rhinitis effectively[5]. Firsteneration antihistamines are associated with sedation, psychomotor retardation, and reduced academic performance. Atopic diseases include allergic rhinoconjunctivitis, asthma, atopic dermatitis, and food allergies, and they tend to run in families. Atopy has been linked to many genetic loci on chromosomes 2, 5, 6. Other risk factors for allergic rhinitis include ethnic origin other than white European, high socioeconomic status, environmental pollution, birth. Allergic rhinitis has been classified traditionally as seasonal or perennial, depending on sensitisation to cyclic pollens or year-round allergens such as dust mites, animal dander, cockroaches, and moulds. This scheme fails globally since seasons do not exist in many areas of the globe, and even where they do, many affected individuals have both seasonal and perennial allergen sensitisation. The roots of allergy are in parasitology, when a nematode parasite attacking the nasal mucosa. On first presentation, the immune

eISSN: 2349-5715 pISSN: 2349-5707

Volume: 12, Issue 01, January-2025 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980 2024: 7,662

system is stimulated to produce immunoglobulin E (IgE) that will bind to and prime mast cells and other inflammatory cells. On a subsequent presentation, nematode antigens interact with the mast cell IgE, causing it to release preformed histamine to make the local environment hostile (e.g. producing mucus and stimulating sensory nerves to cause sneezing). Mast cells also produce cytokines to stimulate the influx inflammatory cells, particularly eosinophils that contain several toxic proteins with which they kill the nematode and cause local inflammation. Patients with allergies mount the same response to allergens as nematode antigen. Patients with allergic rhinitis may also experience ocular symptoms, primarily reddened, itchy and watery eyes. Classically these symptoms were believed to be caused by the allergen landing on the conjunctival lining of the eye, with subsequent activation of the conjunctival mast cell[6]. It is now believed that these symptoms are partly the result of a naso-ocular reflex in which allergic inflammation in the nose stimulates the trigeminal nerve with subsequent release of neuropeptides in the tear. These neuropeptides activate conjunctival mast cells that release histamine but cause little subsequent eosinophil infiltration and allergic inflammation. During periods of high atmospheric levels, pollen impaction on the conjunctiva may induce a more severe form of vernal conjunctivitis in which eosinophil infiltration stimulated allergic inflammation. The late-phase reaction develops over a period of hours after exposure to an allergen. It is characterised by cellular recruitment of basophils, neutrophils, T-lymphocytes, monocytes, and eosinophils, and by the release of multiple mediators, including cytokines, prostaglandins, and leukotrienes, which perpetuate the inflammatory response. This late-phase inflammatory reaction is associated with tissue remodelling, further tissue edema, and the development and perpetuation of nasal congestion, considered by patients to be one of the most troublesome symptoms of AR [7]. As a result of mucosal inflammation, tissues become primed and react more vigorously to allergen exposure. These late-phase reactions and modifications in tissue responsiveness contribute to bronchial hyper-responsiveness. Nasal congestion is the hallmark of the allergic response. This symptom is associated with sleep-disordered breathing, a condition that can have a profound effect on productivity and increased daytime sleepiness . Furthermore, many of the key pathophysiological mediators of AR responses (i.e. histamine, leukotrienes, cytokines, and prostaglandins) play a role in sleep regulation and might be directly involved in this feature of the condition, independent of nasal obstruction. T Helper 2 (Th2) cells activate type 2 responses by stimulating B cells to proliferate and differentiate into plasma cells through the production of Th2 cytokines including IL-4, IL-5, IL-6, and IL-13 [8]. Th2 cells are major contributors of IgE-producing B cells , and Th2 cells play a predominant role in AR pathogenesis. Together with eosinophils and basophils, Th2 cells infiltrate the nasal mucosa tissue, resulting in late phase allergic response [8]. IL-4 is a key cytokine in promoting Th2 differentiation from naïve $CD4^+$ T cells . Th2 cytokines not only enhance inflammatory cell activation but also may deregulate epithelial cell barrier integrity in allergic disease (e.g., AR, eosinophilic esophagitis, asthma, and chronic rhinosinusitis) . The cytokines may also be released within the sinonasal microenvironment including sinonasal epithelial cells, causing increased epithelial cell permeability. This is thought to be due to regulation of transmembrane transcription involved in TJ remodeling where the “tight” barrier properties of TJ proteins are switched to “leaky” properties. Th2 cytokines also hinder the epithelial barrier from resealing which may maintain the inflammation and exposure to inflammatory antigens.Increased prevalence of AR and that it is an incurable disease pose major unmet needs to alleviate these issues. AR comorbidities also confer increased health and socioeconomic burden on AR patients. Moreover, diagnosing AR is more challenging in patients with dual AR (DAR), a recently defined AR phenotype in which the DAR patients display perennial and seasonal allergies-related nasal symptoms, and only allergic to seasonal allergies.

eISSN: 2349-5715 pISSN: 2349-5707

Volume: 12, Issue 01, January-2025 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980 2024: 7,662

References:

1.Pawankar R. Allergic diseases and asthma: a global public health concern and a call to action. World Allergy Organ J. (2014) 7:12. 10.1186/1939-4551-7-12

2.Licari A, Manti S, Ciprandi G. What are the effects of rhinitis on patients with asthma? Expert Rev Respir Med. (2019) 13:503–5. 10.1080/17476348.2019.1604227

3.Bousquet J, Khaltaev N, Cruz AA, Denburg J, Fokkens WJ, Togias A, et al. Allergic rhinitis and its impact on asthma (ARIA) 2008 update (in collaboration with the world health organization, GA(2)LEN and AllerGen). Allergy. (2008) 63(Suppl. 86):S8–160. 10.1111/j.1398-9995.2007.01620.x

4.Mullol J. A survey of the burden of allergic rhinitis in Spain. J Investig Allergol Clin Immunol. 2009;19(1):27–34

5.Canonica GW, Mullol J, Pradalier A, Didier A. Patient perceptions of allergic rhinitis and quality of life: findings from a survey conducted in Europe and the United States. World Allergy Organ J. 2008;1(9):138–44

6.Nathan RA. The burden of allergic rhinitis. Allergy Asthma Proc. 2007;28(1):3–9.

7.Walker S, Khan-Wasti S, Fletcher M, Cullinan P, Harris J, Sheikh A. Seasonal allergic rhinitis is associated with a detrimental effect on examination performance in United Kingdom teenagers: case–control study. J Allergy Clin Immunol. 2007;120(2):381–7.

8.Braido F, Baiardini I, Scichilone N, Musarra A, Menoni S, Ridolo E, et al. Illness perception, mood and coping strategies in allergic rhinitis: are there differences among ARIA classes of severity? Rhinology. 2014;52(1):66–71.