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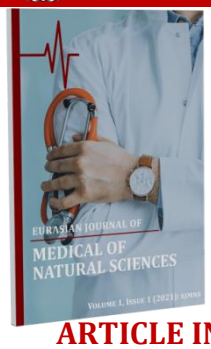

ALLERGIC RHINITIS AND SOME OF ITS FACTORS

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ABSTRACT

The literature review describes the etiology, clinical picture, differential diagnosis and treatment of allergic rhinitis and emphasizes the role of doctors in improving care for patients with this disease.

Allergic rhinitis (AR) is an atopic disease that manifests itself with symptoms of sneezing, nasal congestion, clear rhinorrhea and itching in the nose. It is an IgE-mediated immune response that is directed against inhaled antigens in the immediate phase, followed by a leukotriene-mediated late phase.

АЛЛЕРГИЧЕСКИЙ РИНИТ И ЕГО НЕКОТОРЫЕ ФАКТОРЫ

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ABSTRACT

В обзоре литературы описывается этиология, клиника, диф-диагностика и лечение аллергического ринита и подчеркивается роль врачей в улучшении ухода за пациентами с этим заболеванием.

Аллергический ринит (АР)- это атопическое заболевание, проявляющееся симптомами чихания, заложенности носа, прозрачной ринореи и зуда в носу. Это опосредованный IgE иммунный ответ, который направлен против вдыхаемых антигенов в непосредственной фазе, с последующей лейкотриен-опосредованной поздней фазой.

Introduction. Despite the achievements of modern medicine, allergic rhinitis (AR) is one of the most common allergic diseases, affecting millions of people worldwide. The relevance of the problem is due to many factors. One of which is the increase in the incidence of the disease, the number of patients with allergic rhinitis continues to increase, which is associated with changes in the environment, such as air pollution, climate change and an increase in the number of allergens [24].

Allergic Rhinitis (AR) **is** an atopic condition characterized by multiple symptoms, including nasal congestion, clear rhinorrhea, sneezing, postnasal drip, and nasal itching. It affects approximately one in six individuals and is associated with a high prevalence, leading to

reduced productivity and increased healthcare costs. AR is traditionally considered a pathological process affecting only the nasal airways. However, the development of the unified airway theory has reclassified AR as a component of the systemic allergic response, in conjunction with other comorbid conditions such as asthma and atopic dermatitis, which share a common underlying pathophysiology [10]. Allergic rhinitis can be classified as seasonal (intermittent) or perennial (persistent). Approximately 20% of cases are seasonal, 40% are perennial, and the remaining 40% exhibit symptoms of both types [18]. In addition to nasal symptoms, patients with AR may also experience concomitant allergic conjunctivitis, nonproductive cough, Eustachian tube dysfunction, and chronic sinusitis. Once the diagnosis of AR is established, it is amenable to a variety of treatment strategies. Intranasal corticosteroids remain the first-line therapy due to their effectiveness in controlling inflammation and reducing symptom burden [10].

The etiological mechanisms of the allergic reaction are typically divided into early-phase and late-phase responses. In the early phase, allergic rhinitis represents an IgE-mediated immune response to inhaled allergens, which triggers inflammation driven by T-helper type 2 (Th2) cells [18]. The primary immune response occurs within 5–15 minutes of allergen exposure and results in mast cell degranulation. This process releases a range of mediators, with histamine being one of the key agents involved in the pathophysiology of allergic rhinitis. Histamine induces sneezing via stimulation of the trigeminal nerve, and directly contributes to rhinorrhea by activating mucous glands. Other immune mediators such as leukotrienes and prostaglandins are also involved, acting on blood vessels to cause nasal congestion. Approximately 4–6 hours after the initial response, a cascade of cytokines, notably interleukins IL-4 and IL-13, is released from mast cells, marking the onset of the late-phase response. These cytokines promote the infiltration of eosinophils, T lymphocytes, and basophils into the nasal mucosa, leading to mucosal edema and sustained nasal obstruction [14].

Non-IgE-mediated hypersensitivity may develop as a result of eosinophilic infiltration and obliteration of the nasal mucosa. This leads to heightened sensitivity of the nasal mucosa to common stimuli such as tobacco smoke and cold air, resulting in symptoms like sneezing, rhinorrhea, and nasal itching [12]. There is evidence suggesting a genetic component in the development of allergic rhinitis (AR), although high-quality studies are generally lacking. Monozygotic twins show a concordance rate of 45–60%, whereas dizygotic twins exhibit a rate of around 25%. Certain loci on chromosomes 3 and 4 have also been associated with allergic reactions [20]. The epidemiological prevalence of allergic rhinitis based on physician-diagnosed cases is approximately 15%. However, when considering individuals with nasal symptoms, the estimated prevalence rises to 30%. AR commonly peaks during the second to fourth decades of life and then gradually declines with age [21]. The prevalence of AR among children is also notably high, making it one of the most common chronic conditions in the pediatric population. According to data from the International Study of Asthma and Allergies in Childhood (ISAAC), 14.6% of children aged 13 to 14 and 8.5% of those aged 6 to 7 present with rhinoconjunctivitis symptoms associated with allergic rhinitis [13]. Seasonal allergic rhinitis appears to be more common in children, while chronic rhinitis is more prevalent in adults [2].

A 2023 statistical review indicated that 3.6% of adults missed work and 36% experienced decreased productivity due to allergic rhinitis (AR). Economic analyses suggest that the majority of indirect costs associated with AR stem from loss of work productivity resulting from allergic symptoms [1]. Risk factors for the development of AR include a family history of atopy, male gender, the presence of allergen-specific IgE, serum IgE levels exceeding 100 IU/mL before the age of 6, and a higher socioeconomic status [20]. Studies involving young children have demonstrated a greater risk of AR in those who were introduced to solid foods or formula early and/or were exposed to heavy cigarette smoke during the first year of life [18]. While many studies are exploring the relationship between environmental pollution and the development of AR, a statistically significant correlation has yet to be established. However, several protective factors have been identified. The role of breastfeeding in the development of AR remains under discussion; nevertheless, it continues to be strongly recommended due to its numerous proven health benefits and lack of associated harm. Similarly, there is no definitive evidence that avoiding household pets in childhood prevents the onset of AR. In fact, early exposure to pets may promote immune tolerance. There is growing interest in the so-called "farm effect" on allergy development. In eight meta-analyses, infants raised in rural environments during their first year of life showed a 40% lower risk of developing AR compared to their urban counterparts [22].

There are multiple diagnostic methods available for the assessment of allergic rhinitis (AR). The most common and accessible approach remains a thorough medical history and physical examination. A positive response to empirical treatment with intranasal glucocorticoids may further support the diagnosis. A formal diagnosis can be established via serologic testing for allergen-specific IgE or skin allergy testing [21]. The American Academy of Otolaryngology recommends that allergy testing be reserved for patients who do not respond to empirical therapy or those who require identification of specific allergens for targeted treatment strategies [10]. Serologic testing does not require a trained specialist and does not necessitate discontinuation of antihistamines prior to testing, which can be beneficial for preserving patient quality of life. Skin testing, on the other hand, requires a trained healthcare provider but provides immediate results. To accurately identify triggers in patients with seasonal symptoms, testing should ideally be conducted during peak allergy seasons [21]. Skin prick testing is known to be slightly more sensitive than serologic testing and is generally more cost-effective. Nevertheless, there are contraindications to skin testing, including uncontrolled or severe asthma, unstable cardiovascular conditions, pregnancy, and concomitant use of beta-blockers. In addition, H2-receptor antagonists, tricyclic antidepressants, and monoclonal anti-IgE antibodies such as omalizumab may interfere with skin test reactivity. Therefore, it is recommended that these medications be discontinued prior to testing whenever clinically feasible [22].

Radiographic imaging is generally not recommended for the diagnosis of allergic rhinitis (AR). It is primarily employed to exclude alternative conditions, such as rhinosinusitis [22]. For disease management, trigger avoidance is strongly recommended, particularly in patients with seasonal symptoms, although full avoidance is often challenging. Measures can be taken to reduce exposure to dust mites, animal dander, and upholstered furniture, though these may

require significant lifestyle adjustments, which might not always be acceptable to patients. If removing a household pet is not feasible, restricting the animal to a single room may help reduce allergen exposure. Notably, it may take up to 20 weeks for cat dander to be eliminated from the home environment after the animal's removal. Additional interventions such as using allergen-impermeable bedding, washing bed linens in hot water, and vacuuming with high-efficiency particulate air (HEPA) filters can significantly reduce symptom severity [20]. Pharmacologic treatment options for AR include antihistamines, intranasal corticosteroids, leukotriene receptor antagonists (LTRAs), and immunotherapy. Intranasal corticosteroid therapy may be used either as monotherapy or in combination with oral antihistamines for patients experiencing mild to severe symptoms. Clinical studies have demonstrated that intranasal corticosteroids are more effective than antihistamines in reducing nasal inflammation and improving mucosal pathology.

Intranasal corticosteroids should be considered as the first-line treatment for allergic rhinitis (AR) [23]. Commonly available nasal sprays include beclometasone, budesonide, fluticasone propionate, mometasone furoate, and triamcinolone acetonide. Proper administration of nasal sprays is crucial for achieving optimal clinical response and minimizing side effects. Therefore, patients should always receive counseling on the correct technique for using these medications. Nasal sprays should be used regularly, as their maximum effect may take several days to develop. The spray nozzle should be directed straight into the nostrils and aimed laterally toward the ipsilateral eye to minimize contact with the nasal septum. The most common side effects are nasal irritation and nosebleeds, both of which can be prevented by spraying away from the nasal septum [19]. Oral and injectable steroids have been shown to relieve AR symptoms; however, they are not recommended for routine use due to their significant systemic side effects [22].

First-generation antihistamines include diphenhydramine, chlorpheniramine, and hydroxyzine, while fexofenadine, loratadine, desloratadine, and cetirizine are examples of second-generation antihistamines. Both first- and second-generation antihistamines are effective in controlling the symptoms of allergic rhinitis (AR). However, first-generation antihistamines can be quite sedating due to their ability to cross the blood-brain barrier. These agents also act on muscarinic receptors, leading to side effects such as dry mouth, urinary retention, constipation, and/or tachycardia. Second-generation antihistamines have improved H1-selectivity, are less sedating, and have a longer half-life (ranging from 12 to 24 hours) compared to first-generation antihistamines. Fexofenadine does not cause sedation, but loratadine and desloratadine may cause sedation at higher doses. Cetirizine has the highest potential for sedation among second-generation antihistamines. There is no single superior antihistamine, as all have shown similar efficacy and safety profiles in alleviating symptoms [20]. Topical antihistamines, such as azelastine, have a rapid onset of action and are more effective than oral antihistamines in relieving nasal symptoms. These are recommended as first- or second-line therapy for AR and can be used in combination with intranasal corticosteroid sprays, showing a synergistic effect [22].

Leukotriene receptor antagonists (LTRAs), such as montelukast and zafirlukast, may be useful for patients with allergic rhinitis (AR), but they are not as effective as intranasal

corticosteroids [15]. They are often used in combination therapy with other medications for severe or refractory symptoms. For patients in whom avoidance measures and combined pharmacotherapy are ineffective, allergen immunotherapy should be considered. Subcutaneous immunotherapy (ASIT) or sublingual immunotherapy (SLIT) are commonly used treatment methods. Increasing doses are administered weekly for 6-8 months, followed by a maintenance dose for 3-5 years. Patients typically experience a long-term protective effect, and therapy can be discontinued thereafter [10]. Oral vasoconstrictors, such as pseudoephedrine, are useful for symptom relief but are not recommended for long-term daily use due to their side effect profile. Intranasal vasoconstrictors, such as xylometazoline, are alpha agonists that are directly applied to the nasal tissue, causing vasoconstriction. However, prolonged use of intranasal decongestants carries the risk of rebound nasal congestion (medication-induced rhinitis), so they should not be used for more than one week [22]. Sodium cromoglycate (Cromolyn) is effective in reducing sneezing, rhinorrhea, and nasal itching, making it a reasonable option for treatment. Surgical treatment is intended for patients with nasal polyps, hypertrophy of the inferior nasal turbinates causing refractory nasal congestion, or chronic sinus disease that does not respond to medication [20]. Budesonide is the only approved corticosteroid medication for pregnant patients experiencing allergic rhinitis symptoms [10]. Omalizumab, a monoclonal antibody, is beneficial for patients with AR, although the cost of therapy can limit its use [3]. A nasal saline solution can also be an option when combined with other treatments. Isotonic solutions are helpful for adults, while hypertonic solutions may be more effective for children [22].

The differential diagnosis of allergic rhinitis (AR) involves distinguishing it from other forms of rhinitis that are not allergic, such as vasomotor rhinitis, infectious rhinitis, pregnancy-related rhinitis, hormonal rhinitis, non-allergic rhinitis with eosinophilic syndrome (NARES), chemical rhinitis, drug-induced rhinitis, autoimmune rhinitis, granulomatous rhinitis, vasculitis rhinitis, nasal polyps, nasopharyngeal neoplasms, cerebrospinal fluid leakage, and sickle cell anemia. In the case of young children, especially those under 2 years of age, it is also important to investigate congenital causes of nasal congestion, such as choanal atresia and immunodeficiencies [18, 21, 22].

Complications of Chronic Rhinosinusitis. Although chronic rhinosinusitis differs from allergic rhinitis, it can be a complication of AR. Chronic rhinosinusitis is characterized by inflammation of the nasal passages with symptoms of nasal congestion or discharge lasting for more than 3 months. Chronic rhinosinusitis may also show the presence of nasal polyps (nasal polyposis), which form as a result of chronic inflammation of the sinus mucosa. Nasal polyps are generally benign and tend to occur bilaterally. Unilateral nasal polyps should raise concerns about potential malignancy [24]. The prevalence of nasal polyps in the general population is approximately 4%, with a higher occurrence in men. Treatment options include local steroids and physiological saline irrigation. Surgical removal is reserved for patients who do not respond to medical therapy [10]. Additionally, sensitization to allergens in AR can alter the immunological parameters of the adenoids, potentially leading to adenoid hypertrophy [12]. Eustachian tube dysfunction is commonly seen in patients with AR and presents as ear congestion, otalgia, and ear fullness.

Approximately 10% to 40% of patients with allergic rhinitis (AR) also have a comorbid condition, bronchial asthma, and some studies suggest that asthma is more commonly found in individuals with moderate or severe persistent rhinitis. Numerous studies have demonstrated that AR is an independent risk factor for asthma, particularly in patients who were diagnosed with AR in infancy.

Other associated complications include otitis media with effusion, persistent cough, and eosinophilic esophagitis, although the relationship between these conditions needs further clarification [22].

The provocation of severe exacerbations of rhinitis or asthma, or the development of anaphylaxis, can occur due to allergen desensitization (allergy shots). For this reason, staff working in departments offering such therapy must be well-versed in the diagnosis and management of these severe reactions, and be equipped with appropriate emergency medications (especially epinephrine) and airway management equipment [5, 7].

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