

DOI: 10.5505/anatoljfm.2021.39200 Anatol J Family Med 2022;5(2):68–72

Allergic Rhinitis in Children

Nevin Cambaz Kurt,¹ Durak Ölmez²

¹Department of Pediatry, Basaksehir Cam and Sakura City Hospital, İstanbul, Türkiye ²Sinanpaşa Küçükhüyük Family Health Centre, Afyonkarahisar, Türkiye

ABSTRACT

Allergic rhinitis is one of the most common chronic diseases in all age groups. It is an IgE-mediated allergic inflammation of the nasal airways and is characterized by sneezing, swelling, and inflammation of the nasal mucosa as well as itchy and watery eyes. Laboratory tests used in the diagnosis of allergic rhinitis include allergy skin tests, specific IgE measurements, and imaging studies. The management of allergic rhinitis consists of environmental control, pharmacologic management, and immunotherapy.

Keywords: Allergic rhinitis, children, seasonal allergic rhinitis



Please cite this article as: Cambaz Kurt N, Ölmez B. Allergic Rhinitis in Children. Anatol J Family Med 2022;5(2):68–72.

Address for correspondence:

Dr. Nevin Cambaz Kurt. Department of Pediatry, Basaksehir Cam and Sakura City Hospital, İstanbul, Turkey

Phone: +90 533 372 22 65 E-mail:

nevinbinnurkurt@gmail.com

Received Date: 11.06.2021 Accepted Date: 15.10.2021 Published online: 31.08.2022

Anatolian Journal of Family Medicine - Available online at www.anatoljfm.org



INTRODUCTION

Allergic rhinitis (AR) is an inflammatory disorder of the nasal mucosa induced by allergen exposure triggering an IgE-mediated hypersensitivity reaction.^[1] It is characterized by four major clinical symptoms: nasal obstruction, rhinorrhea, nasal itching, and sneezing.^[2] Itching in the palate, postnasal drip, cough, and poor olfaction are other minor symptoms associated with AR. The presence of a minimum of two of these symptoms which last more than 1 h during the day and are observed for at least two consecutive days suggests AR.^[3] While nasal obstruction might be the only symptom in preschoolers, it is unusual for the nasal obstruction to be observed as the only one symptom^[4] The association between AR and other allergic conditions including rhinoconjunctivitis, asthma, and atopic dermatitis in children is greater than AR in adults.^[5]

Children with AR have observed a picture called allergic salute due to wiping the nose.^[2] A transverse line occurs at the junction between the nasal cartilage and nasal bone due to this movement, which indicates that AR has been present for at least 2 years. Purplish discoloration and edema can be seen in the infraorbital region due to chronic nasal obstruction and venous stasis. On physical examination, the nasal mucosa is edematous and pale. The conchas may be seen as hypertrophic.

AR is the most prevalent chronic respiratory tract disease in children.^[6] The prevalence of AR was very high in the last decade. The worldwide prevalence of AR is 8.5% (0.8-45%), and it has been reported as 2.9%-39.9% in Turkey.^[7]

AR is traditionally subdivided into two groups: seasonal AR (SAR) and perennial AR (PAR).^[4] SAR develops due to grass, tree, and grass pollens; PAR develops due to house dust mites, mold, cockroaches, and the hair of domestic animals.

The patient's sleep quality and daily work activities are normal when the symptoms are mild.^[7] Moderate-severe symptoms include disruption in daily activities and sports as well as poor sleep quality and school performance.

Etiopathogenesis of AR

The allergen is processed by antigen-presenting cells in the nasal mucosa and presented to the T cell receptors of CD4+ T cells in the regional lymph node by MHC class 2 cells after it is ingested by the respiratory tract.^[8] T cells transform into TH2 cells, triggering the release of IgE from B cells through IL-4, IL-5, IL-8, and IL-13. When these IgEs, which bind to the surface of mast cells, are exposed to the allergen again, mediator release is induced. Allergies are triggered by the primary and secondary metabolites that are produced.

Risk Factors for AR

Genetic Factors and Family History: Half of the children with AR have a family member who is atopic.^[8] The presence of allergy in their parents is a significant risk factor. The risk is 60-70% if both mother and father have a history of allergy; the risk is 10% if there is no parental history of allergy.

Early-Life Risk Factors: The early-life risk factors include maternal infection during pregnancy, use of antibiotics during pregnancy, cesarean delivery, perinatal asphyxia, preterm birth, low birth weight, not taking breast milk, use of infant formula, and frequent use of paracetamol in the first year (once a month).^[9,10] The use of antibiotics more than four times a year in infancy increases the risk of AR. The prevalence of AR is increased by frequent upper respiratory tract diseases, tonsillectomy, and low vitamin D levels.

Gender Differences: AR is more common in boys than in girls during childhood, but it becomes more prevalent in girls after adolescence.^[10]

Smoking: Active and passive exposure to cigarette smoke is a significant risk factor.^[11] Cigarette smoke has a cytotoxic effect on sinonasal epithelial cells and inhibits immune function.

Environmental Risk Factors: Air pollution created by motor vehicles in big cities is one of the most important risk factors.^[12] Fuel combustion products affect the immune system by inducing allergic inflammation. It has been reported that exposure to polycyclic aromatic hydrocarbons, which are produced when organic substances such as diesel fuel, wood, coal, and tobacco are burned, cause epigenetic T cell changes, leading to impaired cellular and humoral immunity. Carbon monoxide, nitric oxides, and volatile organic compounds are other environmental risk factors. Formal-dehyde and isocyanates in furniture are also risk factors.^[4]

Indoor Allergens and Indoor Air Pollution: House dust mites, mold antigens, pet hair, insects, scented sprays used at home, and scented cleaning products are factors that increase the risk of AR.^[10]

Risk Factors for Food: Fast food nutrition, consumption of food additives, margarine consumption, and low consumption of fresh food are risk factors.^[12]

Lack of sports activities, gaining weight, insufficient time spent in the fresh air, excessive time spent in front of the television or computer, and sedentary lifestyle are also considered risk factors for children.^[4,10–12]

Although living in a city is a risk factor for the development of AR, it has been stated that living in rural areas has a protective effect on the development of AR.^[13]

Comorbid diseases such as allergic conjunctivitis, recurrent otitis media, sinusitis, adenoid hypertrophy, and asthma are common in children with AR.^[7] Other comorbid conditions include recurrent respiratory tract infections, postnasal drip, nasal obstruction, adenoid and tonsil hypertrophy, nasal polyps, oral breathing, and obstructive sleep apnea.

Children with AR may suffer from sleep disorders, attention deficit, perceptual disorders, decrease in school success, fatigue, depression, and anxiety.^[14]

AR is particularly important because it is frequently accompanied by asthma which is difficult to control.^[15] According to a study, it was reported that asthma accompanied 88.6% of patients with PAR and 54.8% of patients with SAR.

It was reported that 36% of the patients with AR were accompanied by depression during the allergy season.^[15] It has been reported that 28.4% of patients with attention deficit hyperactivity disorder have AR symptoms, and the skin prick test is highly positive.^[16]

AR Diagnosis

AR is diagnosed with clinical findings. Laboratory findings are helpful in diagnosis.^[4] Eosinophils are counted in nasal smear with Hansel stain and account for more than 4% has been considered significant. Specific IgE levels can be measured in the blood.^[7,8] The skin prick (or also called scratch) allergy test is the most important diagnostic method.^[4] Antihistamines should be stopped 10 days and methylprednisolone 4 weeks prior to allergy testing as they will affect the results.^[8]

AR Treatment

In AR treatment, avoidance of triggering factors is the first treatment, while drugs are the second treatment.^[7] AR

treatment is a long-term treatment.^[8] Symptoms cannot be controlled in 20% of patients, and asthma develops in 10%-20% within 5-10 years.

Avoidance of Trigger Factors: Because house dust mites are the most common cause of AR, precautions should be taken against mites.^[17] However, measures against house dust mites alone will not suffice to reduce AR symptoms. Special anti-dust mite mattress covers reduced bed mites by 30% when compared with placebo; however, it has been demonstrated that its clinical effect is insufficient. HEPA filters have not been effective in reducing nasal symptoms. ^[4,8] When the precautions against allergens are taken as a whole, integrated measures such as removing carpets in the bedroom, washing all walls and floors, frequently changing duvets and pillows and washing at high temperatures, washing every 2 weeks if there is a pet in the house, and avoiding smoking exposure were found to be more effective. In the presence of pollen allergy, it may be advised to keep the windows closed particularly in the morning hours, to wear hats and sunglasses when going outside, and to limit the amount of time spent outside during the pollen season.^[4]

Medical Treatment: Oral medications are used for the treatment of AR.^[4,7,18,19] The efficacy of these medications varies from person to person. Intranasal corticosteroids (INCS) administration is the most effective treatment.

- 1. Intranasal glucocorticosteroids: They are the most effective medications for allergic and non-AR. It is effective for all rhinitis symptoms. It is the first option for nasal obstruction.^[4,7,8] It is also effective on eye symptoms.^[7] Its effectiveness begins after 7-8 h, maximum beneficial effects manifest themselves after 2 weeks. It should be used prior to the season in seasonal symptoms.^[8] Fluticasone furoate is used in children above 2 years, mometasone furoate above 3 years, budesonide above 6 years, and fluticasone propionate and beclomethasone above 12 years.^[8] INCS are well tolerated, have no systemic absorption, and no systemic adverse effects. Adverse effects are rare and mild. A slight slowdown in height growth was found in patients who used intranasal beclomethasone for 1 year.[20] This effect was not observed with other nasal steroids.[21]
- Oral antihistamines: Effective against nasal drip, itching, sneezing, and eye symptoms, they have little effect on nasal obstruction.^[4] Long-term treatment with oral antihistamines is safe.^[8,15] First-generation oral antihistamines cause sedation; this feature is absent in second-generation antihistamines. Some antihistamines

may interact with macrolide antibiotics and antifungal drugs. Arrhythmia has been reported with terfenadine, astemizole, and diphenhydramine. Caution should be exercised in patients with long QT Syndrome.^[15] Intranasal antihistamines such as azelastine are not as effective as steroids in relieving itchy nose, sneezing, and rhinor-rhea.^[16] It is not recommended for use in the treatment of PAR.^[15]

- 3. Leukotriene receptor antagonists: It is effective against nasal congestion, rhinorrhea, and sneezing symptoms. Montelukast has been demonstrated to reduce the need for β-agonist use in patients with SAR and asthma.^[7] It is more effective than a placebo in the treatment of SAR, the same as oral antihistamines, and weaker than INCS.^[4] Studies showed no clinically significant difference between the effect of montelukast and the oral antihistamine loratadine. Montelukast-loratadine combination is not more effective than single use. Montelukast-cetirizine combination was found to be beneficial when started 6 weeks before the pollen season. Montelukast causes hallucinations in some children.^[8]
- 4. Decongestants: Intranasal decongestants act by vasoconstriction in nasal congestion seen in allergic and non-AR.^[18] However, a runny nose and an itchy nose are not effective in sneezing symptoms. Its effect lasts 12 h in topical use and 24 h in oral use. They are not used for more than 10 days. They can cause tachyphylaxis, swelling of the nasal mucosa, and drug-induced rhinitis. ^[4] Systemic side effects such as restlessness, insomnia, headache, hypertension, and tachycardia can be seen in oral intake. In meta-analyses, it has been reported that nasal irrigation with saline is beneficial in reducing nasal secretion and in the treatment of AR with its mild decongestant effect.^[19]
- 5. Chromolins: The effect of these mast cell stabilizers are limited in intranasal use.^[15]
- 6. Immunotherapy: The effect of immunotherapy in AR treatment has been demonstrated in double-blind, placebo-controlled studies.^[4,22,23] It has been reported that subcutaneous immunotherapy against a single allergen protects against new sensitizations. It is claimed that it has an effect on preventing the development of asthma in patients with AR. The effect of subcutaneous immunotherapy has been shown in both asthma and AR.^[4] The optimal dose to be administered should have a clinical effect without causing side effects. Immunotherapy is not recommended for those below 5 years.^[15]

Sublingual immunotherapy is especially recommended for pollen allergies. In some studies, it has been shown to be safe in children below 5 years.^[7] Its adverse effects are rare.^[4,23]

CONCLUSION

AR is a common disorder that occurs in children and adolescents. The disease is associated with other allergic diseases, such as asthma, and it carries a heavy burden, with effects on sleep, school performance, and quality of life. Treatment includes patient education, irritant or allergen avoidance, and pharmacotherapy.^[24] The most frequently used pharmacological treatments include oral, intranasal antihistamines, INCS, or a fixed combination of intranasal H1-antihistamines and corticosteroids.^[25] Allergen immunotherapy prescribed by a specialist using high-quality extracts in stratified patients is effective in patients with persistent symptoms. Allergic rhinitis should be kept in mind in the differential diagnosis in repetitive serous otitis media, especially in childhood.^[26]

Disclosures

Peer-review: Externally peer-reviewed.

Conflict of Interest: The authors declare that there is no conflict of interest.

Funding: The author received no financial support for the research, authorship, and/or publication of this article.

Authorship Contributions: Concept – N.C.K., B.Ö.; Design – N.C.K., B.Ö.; Supervision – N.C.K., B.Ö.; Materials – N.C.K., B.Ö.; Data collection and/or processing – N.C.K., B.Ö.; Analysis and/or interpretation – N.C.K., B.Ö.; Literature search – N.C.K., B.Ö.; Writing – N.C.K., B.Ö.; Critical review – N.C.K., B.Ö.

REFERENCES

- Skoner DP. Allergic rhinitis: definition, epidemiology, pathophysiology, detection, and diagnosis. J Allergy Clin Immunol 2001;108(1 Suppl):2–8. [CrossRef]
- Brożek JL, Bousquet J, Agache I, Agarwal A, Bachert C, Bosnic-Anticevich S, et al. Allergic rhinitis and its impact on asthma (ARIA) guidelines-2016 revision. J Allergy Clin Immunol 2017;140(4):950–8. [CrossRef]
- International Rhinitis Management Working Group. International Consensus Report on Diagnosis and Management of Rhinitis. Allergy 1994;49(Suppl 19):1–34. [CrossRef]
- 4. Güler N. Çocuklarda alerjik rinitin tanı ve tedavisi: pratik ve güncel yaklaşım. Klinik Tıp Pediatri Dergisi 2017;9(6):28–40.
- Dominguez IA, Jauregui I, Del Cuvillo A, Montoro J, Davila I, Sastre J, et al. Allergy rhinitis: similarities and differences between children and adults. Rhinology 2017;55(4):326–31.
- 6. Jáuregui I, Dávila I, Sastre J, Bartra J, del Cuvillo A, Ferrer M,

et al. Validation of ARIA (Allergic Rhinitis and its Impact on Asthma) classification in a pediatric population: the PEDRIAL study. Pediatr Allergy Immunol 2011;22(4):388–92. [CrossRef]

- 7. Özdemir Ö, Elmas B. Alerjik rinitte yenilikler. Asthma Allergy Immunol 2016;14:1–16. [CrossRef]
- Öneş Ü. Alerjik rinit. Pediyatri cilt 2. In: Neyzi O, Ertuğrul T, Darendeliler F, editors. 5th ed. Ankara: Nobel Tıp Kitabevi; 2021. p. 923–27.
- 9. Alm B, Goksör E, Pettersson R, Möllborg P, Erdes L, Loid P, et al. Antibiotics in the first week of life is a risk factor for allergic rhinitis at school age. Pediatr Allergy Immunol 2014;25:468–72.
- Özdemir Ö, Elmas B. Alerjik rinitin değişken prevalansı ve prevalansı etkileyen risk faktörleri. Kulak Burun Bogaz Ihtis Derg 2016;26(6):371–82. [CrossRef]
- Higgins TS, Reh DD. Environmental pollutants and allergic rhinitis. Curr Opin Otolaryngol Head Neck Surg 2012;20(3):209– 14. [CrossRef]
- 12. Sultész M, Horváth A, Molnár D, Ktona G, Mezei G, Hirschberg A, et al. Prevalence of allergic rhinitis, related comorbidities and risk factors in schoolchildren. Allergy Asthma Clin Immunol 2020;16(1):98. [CrossRef]
- 13. Leynaert B, Neukirch C, Jarvis D, Chinn S, Burney P, Neukirch F. Does living on a farm during childhood protect against asthma, allergic rhinitis, and atopy in adulthood?. Am J Respir Crit Care Med 2001;164(10 Pt 1):1829–34. [CrossRef]
- 14. Munoz-Cano R, Ribo P, Araujo G, Giralt E, Sanchez-Lopez J, Valero A. Severity of allergic rhinitis impacts sleep and anxiety: results from a large Spanish cohort. Clin Transl Allergy 2018;8:23. [CrossRef]
- 15. Duman H, Mısırlıoğlu ED, Giniş T, Bostancı İ. Çocuklarda alerjik rinit. Çocuk Dergisi 2010;10(2):62–8. [CrossRef]
- 16. Yanez A, Rodrigo GJ. Intranasal corticosteroids versus topical H1 receptor antagonists for the treatment of allergic rhinitis: a systematic review with meta-analysis. Ann Allergy Asthma Immunol 2002;89(5):479–84. [CrossRef]
- 17. Sheikh A, Hurwitz B, Shehata Y. House dust mite avoidance measures for perennial allergic rhinitis. Cochrane Database Syst Rev 2007;24(1):CD00156. [CrossRef]
- Green RJ, Van Niekerk A, McDonald M, Friedman R, Feldman C, Richards G. Acute allergic rhinitis. S Afr Fam Pract (2004) 2020;12;62(1):1–6. [CrossRef]
- 19. Bernstein DI, Schwartz G, Bernstein JA. Allergic rhinitis: mechanisms and treatment. Immunol Allergy Clin North Am 2016;36(2):261–78. [CrossRef]
- Spector SL, Nicklas RA, Chapman JA, Bernstein IL, Berger WE, Moore JB, et al. Symptom severity assessment of allergic rhinitis: part 1. Ann Allergy Asthma Immunol 2003;91(2):105–14.
- 21. Allen DB, Meltzer EO, Lemanske RF Jr, Philpot EE, Faris FA, Kral KM, et al. No growth suppression in children treated with the maximum recommended dose of fluticasone propionate aqueous nasal spray for one year. Allergy Asthma Proc

2002;23(6):407-13.

- 22. Schuler Iv CF, Montejo JM. Allergic Rhinitis in children and adolescents. Immunol Allergy Clin North Am 2021;41(4):613–25.
- 23. Calderon MA, Alves B, Jacobson M, Hurwitz B, Sheikh A, Durham S. Allergen injection immunotherapy for seasonal allergic rhinitis. Cochrane Database Syst Rev 2007;24;2007(1):CD001936.
- 24. Brown T. Diagnosis and Management of Allergic Rhinitis in Children. Pediatr Ann. 2019;48(12):e485–8. [CrossRef]
- 25. Bousquet J, Anto JM, Bachert C, Baiardini I, Bosnic-Anticevich S, Walter Canonica G, et al. Allergic rhinitis. Nat Rev Dis Primers 2020;3;6(1):95. [CrossRef]
- 26. Scheinmann P, Pham Thi N, Karila C, de Blic J. Marche allergique chez l'enfant, de la rhinite à l'asthme : prise en charge, place de la désensibilisation [Allergic march in children, from rhinitis to asthma: management, indication of immunotherapy]. Arch Pediatr 2012;19(3):330–4.