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Review Article

Allergic Rhinitis in Children

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Abstract

Alex, a 7 year-old Caucasian male, is brought to the primary care clinic by his mother for a routine well-child check. In obtaining the history the Nurse Practitioner (NP) is made aware of increasing issues with snoring, nasal congestion, ophthalmic itching, frequent sore throat upon wakening, and afternoon fatigue. Home treatment includes over the counter (OTC) cetirizine (Zyrtec) 10mg daily for the last month with minimal improvement, mother reports the medication increases afternoon fatigue and difficulty concentrating at school. Alex has no previous significant medical history, is up-to-date on immunizations, and has no other complaints. Alex's father had significant asthma and atopic dermatitis as a child. Physical exam reveals a normally developing Tanner 1 school-aged male, who is appropriate in cognition and use of fine and gross motor skills. Tympanic membranes are clear. Palpebral conjunctiva mildly injected bilaterally. The ophthalmic exam is otherwise unremarkable. Nares are patent, but inferior and medial turbinate's are pale and mildly edematous with scant to moderate clear rhinorrhea. Pharynx with mild cobble stoning and noted clear posterior drainage. Tonsils+2/4 without erythema or drainage. Neck is supple without lymphadenopathy. Heart rate is 88 beats-per-minute. S1, S2 noted and regular without murmur. Lungs clear to auscultation throughout, respirations even and unlabored. The remainder of the physical exam is unremarkable.

Allergic Rhinitis

Allergic rhinitis (AR) is a frequent, yet under treated allergic disease and is one of several common presentations of atopy, the "genetic tendency to develop allergic diseases" [1]. Prevalence of AR is higher in industrialized nations and higher socioeconomic areas due to poor home ventilation, increased environmental pollutants, particularly tobacco and traffic pollutants, dust mites, molds, decline in physical activity, and changes in diet [2,3]. Over 60 million Americans suffer from AR, 10% to 30% of adults, and 10% of children [4-7]. AR affects boys (10.6%) more often than girls (8.6%), Caucasian children (10.3%) more than African American (6.6%) or Hispanic (7%) children, is highest in prevalence during the adolescent years, and lowest in infants to four year olds [3]. The economic impact of AR is estimated to be $3.4 billion in direct medical costs per year and as many as 2 million missed school days annually [5].

AR is reversible, non life threatening, and was previously considered clinically insignificant. However, the relationship of AR to the spectrum of allergic disease, quality of life, and disease burden are significant enough to warrant treatment in affected children. Patients with AR typically average more prescription medications and office visits per year than other patients, yet as many as 1/3 of children suffering from AR never visit a health care provider regarding treatment of the condition [8,9]. The purpose of this article is to provide an overview of AR and its treatment in pediatric patients.

Background

Rhinitis can be allergic or non-allergic (Table 1). AR is an Immunoglobulin-E (IgE) moderated inflammatory disorder of the upper respiratory and nasal passageways characterized by sneezing, itching, rhinorrhea, and/or congestion [6,10]. Diagnosis of AR typically is not made prior to age 2, with peak diagnosis between the ages of 6 to 8, and 80% of cases diagnosed prior to age 20 [5,10,11]. However, children younger than two can present with symptoms, but there is difficulty differentiating AR from infectious upper respiratory infections (URIs), common in younger children [2]. Family history of allergic disease, environmental exposures, and presence of other atopic diseases along with rhinitis increases the likelihood of allergic causes.

Previously known as perennial or seasonal allergic rhinitis (PAR, SAR), AR is now classified as persistent (PAR) or intermittent (IAR) by the Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines [9,12,13]. Classification is a two-step process including persistence and severity of symptoms as either mild or moderate/severe [11-13].

Children with AR are more than twice as likely to be diagnosed with asthma, visit the emergency room for an allergic associated disease, and three times more likely to experience hospitalization for reactive airway disease [14] .They are more irritable and fatigued, less attentive in school, and may be more likely to be socially secluded [3]. Children with AR are also less likely to be perceived as having

Table 1: Triggers of non-allergic rhinitis.

- Infectious diseases
- Temperature changes
- Changes in weather
- Foreign bodies
- Choanal atresia
- Polyps/tumors
- Septal wall defects
- Pregnancy
- Hormonal contraceptives
- Nonsteroidal anti-inflammatories
- Emotion
- Chemicals
- Odors

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'excellent health' by parents, less happy, less peaceful, and to have diminished school performance [3]. Teenagers using antihistamines for AR have lower exam scores [9].

Risk Factors

Factors associated with the development of AR include early introduction to foods or formula in infancy, maternal smoking at < 1 year of age, serum IgE level > 100 Iu/ml before 6 years of age, and food allergies [10,15]. AR is on of several conditions present in atopic patients. The trifecta of atopy includes eczema, AR, and reactive airway disease. Co-morbidities for children with AR include sinusitis, otitis media, increased frequency of upper respiratory infections, and conjunctiva symptoms [5,9,10,15]. The most common triggers for AR are pollen (54%), dust mites (29%), and animal dander (15%) [3].

The most frequent pollen triggers are tree in the springtime, grasses in the summer, and weeds in the fall, but these can vary depending on geographic location. Molds are fungal sources that grow best in moisture and can be indoor or outdoor. Dust can trigger reaction, but dust mites are a common trigger. Dust mite excrement causes eosonophilic reaction when inhaled. Humidity increases the dust mite count and they live abundantly in places with a good supply of skin cells, their primary food source, such as mattresses, pillows, carpet, and on animals. Animal dander triggers are the proteins found in skin and saliva cells. For example, feline saliva and skin cells carry triggering proteins [17].

Pathophysiology

Rhinitis develops from exposure to protein antigens and triggering of nasal mucosa with subsequent swelling and increased secretions [5,10,16]. Repeated antigen exposure creates allergic sensitization through IgE, mast cell, and T-lymphocyte immune responses. Mast cells in the nasal mucosa degranulation and release chemotactic mediators of inflammation such as histamine and leukotriens when IgE specific to a sensitized allergen on the mast cell surface binds with that allergen [16,18].

Histamine receptors are present in nerve, airway, vascular smooth muscle, endothelial, and epithelial cells, and when activated cause pruritus, pain, vasodilation, vascular permeability, hypotension, flushing, headache, tachycardia, bronchoconstriction, cough, and airway vagal afferent nerve stimulation. Mast cells and basophils store histamine and release large quantities into nasal mucosa when IgE molecules and allergens bind. Histamine's vasodilation and vasopermeability leads to rhinorrhea and congestion. Histamine also mediates a parasympathetic reflex that stimulates glandular secretions compounding rhinorrhea. Sneezing and itching of the nose, palate, and throat occurs as histamine stimulates sensory nerves of the upper airway [19]. As cells become inflamed, neutrophil, eosinophils, lymphocytes, monocytes/macrophages, and dendric cells migrate to the area of inflammation and contribute to the late phase symptoms in which congestion dominates [19,20].

Genetic tendencies for atopy are strong factors for developing disease and several genetic polymorphisms have been linked to allergic disease, with several phenotypes noted on more than 14 pairs of chromosomes. Further, Type 2 helper T cells (Th2) assist in the activation of B cells that produce IgE antibodies. Reduced exposure to a variety of environmental microbes early in life causes the immune system's immature CD4 T-lymphocytes to create more Th2 cells instead of Th1 cells. Th2 cells secrete several substances, one of which is IL-4. Th1 cells secrete interferon-gamma (INF-γ). IL-4 and INF-γ, among other cytokines, counter-regulate each other. Reduced Th1 production reduces INF-γ and creates an imbalance between cytokines, leading to greater tendencies for allergic disease. The balance of Th1 to Th2 cells is important because Th1 cells inhibit IgE, where IL-4 stimulates IgE production [17].

Clinical Manifestations

Historical symptoms of AR are similar to URI and include nasal congestion, frequent or repeated sneezing, runny nose, and watery eyes [3]. Children with AR may also experience frequent nose blowing, sniffing, snorting, nasal rubbing, itching, picking the nose, and snoring. Other symptoms include the allergic gape (open-mouth breathing), ocular itching, otalgia, nasal salute (wiping of the nose in an upward palmar fashion) and possibly headache or fatigue may also be noted by children or parents. These symptoms differ from upper respiratory infection (URI) symptoms in the length of duration, color of drainage, and symptoms that typically accompany a URI such as myalga and fever.

Physical exam findings include clear rhinorrhea, conjunctiva irritation, eye watering, eyelid edema, nasal crease, allergic shiners (dark circles under the eye), dental malocclusion, dennie-morgan lines under the lower eye lid due to chronic swelling, middle ear effusion, swelling and pallor of nasal turbinate's, and cobble stoning of pharynx or conjunctiva [5,6,9]. Physical exam findings do not always correlate with severity of disease and rate poorly on guidelines as a diagnostic indicator of AR.

Laboratory Testing

Serum testing of elevated Ig-E antibodies through IgE immune CAP testing can be used for first line screening and when coupled with symptoms and atopic disease are highly suggestive of allergic rhinitis [7,19]. Immuno CAP testing is an immunoassay that measures level of Ig-E to specific allergens such as dust mites, foods, medications, or pollens. However, if treatment should be the first approach when AR is suspect as testing is not specific and is costly. If treatment is not effective, referral to an allergen for specific IgE skin testing is advised.

Treatment

Symptoms of AR will not cause long-term damage and the condition is not life threatening. However, treating the disorder can diminish nasal symptoms and improve quality of life. Controlling AR can diminish asthma symptoms and exacerbations and diminish the incidence of sinus infections, thus improving quality of life [19,22]. Treatment of symptoms is dependent on severity of symptoms and impact on daily activities, including sleep. An overview of medication options is presented in Table 2. Treatment options include avoidance of known allergens (environmental control), intranasal and oral corticosteroids, oral and intranasal antihistamines, oral and topical decongestants, leukotriene antagonists, intranasal cromolyn, intranasal anticholinergics, and nasal saline rinses. Additionally, specific allergen immunotherapy is used in severe cases of AR.

Environmental control is considered first line treatment in both Allergic Rhinitis and Its Impact on Asthma (ARIA) and American

Table 2: Common Drugs Used for Allergic Rhinitis.

Drug Class	Examples	Approved for Use	Mechanism of Action	Adverse Effects
Intranasal Corticosteroids	Budesonide (Rhinocort Aqua) Fluticasone (Flonase propionate) Mometasone (Nasonex)	6 years and older 4 years and older 2 years and older	Prevent inflammatory response to allergens reducing all symptoms of AR	Nasal Irritation
Oral Antihistamines (First Generation-Sedating)	Chlorpheniramine Diphenhydramine	6 years and older Dosing guidelines for less than 6 years	Block H_1 receptors to decrease itching, sneezing, and rhinorrhea. No reduction in nasal congestion	Sedation and anticholinergic effects
Oral Antihistamines (Second-Generation-Non Sedating)	Cetirizine*(Zyrtec) Levocetirizine* (Xyzal) Fexofenadine (Allegra) Loratadine (Claritin) Desloratadine (Clarinex)	6 years and older 6 years and older 12 years and older 6 years and older 12 years and older	Block H_1 receptors to decrease itching, sneezing, and rhinorrhea. No reduction in nasal congestion	Cetirizine and levocetirizine can cause sedation
Intranasal Antihistamines	Azelastine (Astelin) (Astepro) Olopatadine (Patanase)	5 years and older Astepro 12 years and older 12 years and older	Block H1 receptors to decrease itching, sneezing, and rhinorrhea; do not reduce congestion	
Intranasal Cromolyn	NasalCrom	2 years and older	Prevents release of inflammatory mediators from mast cells	None
Oral Decongestants	Pseudoephedrine (Sudafed)	Dosing guidelines for less than 6 years	Activate vascular alpha$_1$ receptors causing vasoconstriction which reduces nasal congestion. No effect on itching or sneezing	Restlessness, insomnia, increased blood pressure
Topical Nasal Decongestants	Oxymetazoline (Afrin)	6 years and up	Activate vascular alpha$_1$ receptors in the nose causing vasoconstriction which reduces nasal congestion. No effect on itching or sneezing	Rebound nasal congestion (rhinitis medicamentosa)
Leukotriene Antagonists	Montelukast (Singulair)	12 months and older	Blocks binding of leukotrienes to their receptors to relieve nasal congestion. Has little effect on sneezing and itching	Neuropsychiatric effects (agitation, aggression, depression, insomnia)
Intranasal Anticholinergic Agents	Ipratropium (Atrovent Nasal spray)	12 years and older	Blocks cholinergic receptors to inhibit glandular secretions and decrease rhinorrhea	Nasal drying and irritation

*May cause some sedation at recommended does.

College of Allergy, Asthma, and Immunology (ACAAI) Guidelines and is the safest, most economical, and easiest method for decreasing AR symptoms [4,9,11,12]. Avoiding pollens, fungi, dust mites, cockroaches, animals and other irritants is beneficial in controlling AR [19].

Updates to ARIA guidelines do not include recommendation for expensive dust-mite prevention through chemicals or barrier covers. Instead, thorough vacuuming, use of non-carpeted floors, and control of environmental humidity to decrease dust mite and mold activity is encouraged. Removal of household pets is also no longer recommended, but limiting exposure, such as not allowing the pet to sleep with the child with AR, is recommended [9,10]. Other easy steps that parents can take includes showering after playing outside and prior to bedtimes to prevent children from depositing pollen into pillows and fabric, keeping widows closed, particularly in the room the child is sleeping in, and removal of environmental chemicals, odors, or triggers such as tobacco or wood smoke [9,10].

Prevention of allergy development is of priority, particularly for high-risk children with a strong family history of atopy and allergic disease. Exclusive breast-feeding for at least three months for all children and avoidance of environmental tobacco smoke for all children and pregnant women is recommended [11,12].

Intranasal corticosteroids are the most effective treatment for AR because they have the ability to block most of the inflammatory mediators that cause the symptoms associated with AR [20]. In addition to blocking inflammatory agents, Glucocorticoid suppresses infiltration of phagocytes to avert damage from lysosomal enzymes that cause cell damage. Corticosteroids also suppress migration of lymphocytes that contributes to inflammation when the allergic response occurs [23]. Just as inhaled corticosteroids are the most effective treatment for persistent asthma, intranasal use of corticosteroids, because they block the majority of the inflammatory mediators out performs agents that block only a single inflammatory substance such as histamine, or leukotrienes.

Oral corticosteroids are sometimes recommended in severe cases of AR for a short burst of 5-7 days [20]. The mechanism of action in the allergic response is the same as the intranasal corticosteroids with the added threat of adverse effects with the use of systemic Glucocorticoid.

Oral antihistamines act as inverse agonists by binding and stabilizing histamine receptors. They are less effective than intranasal corticosteroids in reducing nasal symptoms because they block histamine, only one of the inflammatory agents released by the mast cell [23]. There are 40 oral antihistamines currently on the market with structural similarity. They can be divided into older first generation agents that tend to be sedating and newer second-generation agents preferred due to less sedation (See Table 2).

First generation oral antihistamines readily cross the blood-brain barrier because they are lipophilic and have a low molecular weight. The P-glycoprotein efflux pump in the CNS does not recognize older antihistamines and thus their concentrations in the CNS are significant. Even nighttime administration of older agents can result in sedation and impaired performance the morning after ingestion. In addition, first generation antihistamines bind to muscarinic

cholinergic, α-adrenergic, and serotonergic receptors creating the classic anticholinergics side effects of dry mouth, urinary retention, constipation, and tachycardia.

Second generation oral antihistamines are preferred in the treatment of AR. New agents are metabolites of older antihistamines and have more favorable pharmacokinetics such as better absorption, longer half-life, and decreased effects on the CNS. In addition, second generation antihistamines have rigorous clinical trials demonstrating efficacy in decreasing symptoms of AR as opposed to older agents. Both cetirizine and levocetirizine are classified as second-generation antihistamines, however, both are considered sedating and prohibited for people in certain occupations that demand high psychomotor skills such as flying planes and operating heavy machinery. Cetirizine, levocetirizine, and fexofenadine have the added benefit of diminishing symptoms of allergic conjunctivitis and nasal congestion, not typically a feature of oral antihistamines [19]. The remaining second generation antihistamines are not sedating when used at recommended doses.

Cetirizine, levocetirizine, and desloratadine have been approved for children as young as 6 months of age. Loratadine and fexofenadine have been approved for children as young as 2 years of age. Cetirizine, levocetirizine and loratadine are FDA pregnancy category B and fexofenadine and desloratadine are category C. Fexofenadine, loratadine, and desloratadine are considered safe during lactation [22]. Adding an oral antihistamine with intranasal Glucocorticoid does not provide added benefit to the use of intranasal steroid alone, but it may improve ocular symptoms [19,20].

Intranasal antihistamines are advantageous because they deliver higher concentrations of drug with less systemic side effects, have a rapid onset of action (30-150 minutes-as compared to eight hours with mometasone), and are considered equal to or superior to oral second-generation antihistamines [19]. In addition, they have a clinically significant effect on nasal congestion and a potential benefit for ocular allergic symptoms. Azelastine (Astelin) has an added indication for vasomotor rhinitis; however, the competing agent olopatadine (Patanase) has less of a bitter taste. Somnolence can be avoided by using 1 rather than 2 sprays daily [19]. Astelin, pregnancy category C and is approved for use in children 5 years and older. Efficacy in both is similar. However, nasal corticosteroids remain more effective. The best approach in patients who do not respond to monotherapy may be a combination intranasal corticosteroid and intranasal antihistamine [19].

Oral and topical decongestants are alpha-adrenergic receptor agonists (sympathomimetics) used for AR as well as the common cold, sinus infections, and pressure pain during travel. Decongestants reduce inflammation via vasoconstriction, but have many side effects that limit their use on a regular basis including hypertension, insomnia, and loss of appetite, irritability, palpitations, and hyperactivity. Topical decongestants like Afrin have little systemic involvement and if used as directed (3 days or less). Overuse can lead to the rebound congestion. In general, over-the-counter cold medications such as decongestants, antihistamines, and antitussives in young children are not effective, not recommended, and can be toxic [20,23].

Leukotriene antagonists such as montelukast (Singulair) are equal in efficacy to oral antihistamines in the treatment of AR and are not as effective as nasal corticosteroids [19]. Leukotrienes are released by mast cells and behave similarly to histamine. Some individuals have a larger leukotriene component in their allergic response and therefore have a greater therapeutic effect with these more expensive agents. Because leukotriene antagonists are also indicated for treatment of asthma, using them in patients with this comorbidity can be helpful. New precautions about mood and behavioral changes for patients taking leukotriene modifiers include insomnia, agitation, anxiety, and depression [24].

Intranasal anticholinergics such as ipratropium (Atrovent) nasal spray reduces rhinorrhea but does not have an effect on the other symptoms of AR**.** Drying of the mucous membranes occurs due to the anticholinergics effects. It can be effective in non-allergic rhinitis such as cold-induced rhinitis, and gustatory rhinitis that occurs after eating [20].

Intranasal cromolyn is an older medication available as a nasal spray for treatment of AR. Cromolyn suppresses inflammation by stabilizing the cytoplasmic membrane of mast cells and blocking histamine. The onset of action is rather slow (4-7 days) and is best used in anticipation of allergen exposure [20]. Chromolyn is also available in as an ophthalmic solution for the treatment of allergic conjunctivitis.

Subcutaneous Immunotherapy is the only treatment that creates remission of atopic diseases. Environmental control and medication therapy are first line treatments, but for patients who have continued refractory disease despite therapy, associated atopic diseases, and poor quality of life, subcutaneous immunotherapy is an option. Such therapy is safe and effective when given to patients who have been fully screened for specific allergens and in clinical settings equipped for administration and possible side effects, which include anaphylaxis [26]. Subcutaneous immunotherapy is recommended for those. Sublingual immunotherapy has not been approved by the FDA, but is being used for allergic disease and shows good promise in symptom control [27].

Conclusion

AR is a frequent and costly disorder in the pediatric population. AR can affect quality of life, is commonly present with other allergic, respiratory, and atopic diseases, and is frequently undertreated by health care providers. Nurses serve a pivotal role in recognition, education, and assisting families in disease and medication management. Advance practice nurses can improve treatment rates among patients who suffer from AR. Diagnosis is made through history and symptom presentation. The most accurate way to identify allergen triggers is through skin testing. However, less invasive testing can also be helpful in disease recognition and management. Such testing can follow symptom treatment if treatment failure or persistence of symptoms remains. First line management of children with AR is environmental manipulation and removal of allergens. Intranasal corticosteroids offer the best treatment options and can be combined with other medications for improved effect. Failure of environmental controls plus medication management, more than one medication or combination medications, or persistent symptoms should trigger consideration of referral to the allergy specialist.

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