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Rhinitis

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KEYWORDS

• Rhinitis • Allergy • Antihistamine • Nasal congestion

KEY POINTS

- Seasonal allergic rhinitis is commonly caused by a variety of pollen allergens. Perennial allergic rhinitis is mainly caused by dust mites and animal dander. Occupational rhinitis results from airborne particles in the workplace and is a response to allergic and nonallergic mechanisms.
- Mild disease can usually be managed with avoidance measures alone. Allergen removal can also improve the severity of allergic rhinitis and can reduce the need for medications. Pollen, dust mites, animals, insect proteins, and fungi are the most prominent allergic triggers. Environmental control may take weeks to months to produce full beneficial effects, and complete avoidance of allergen is usually not feasible or practical.
- Allergic rhinitis is represented by sneezing, nasal congestion, nasal pruritus, and rhinorrhea. When allergic disease is suspected, a skin prick test can be performed for confirmation. When skin testing is difficult to interpret or not feasible (ie, dermatographism), allergen-specific immunoglobulin (Ig) E serum testing based on the patient's history can be useful. The specific in vitro assays chosen should be based on local prevalence of aeroallergens.
- Oral antihistamines should be used to treat patients with mild or occasional seasonal allergic rhinitis. Cromolyn is an alternative to oral antihistamines in mild disease.
- The pathophysiology of nonallergic rhinitis does not involve IgE mediation. The specific mechanism is poorly understood because of the many presentations of the conditions in this category. What is known about the pathophysiology is that the nonallergic rhinitis is caused by nasal hyperactivity to nonimmunologic stimuli, but little is known regarding the exact mechanism of this nasal hyperactivity.
- Because of the variance in causes of nonallergic rhinitis, treatments also vary. Vasomotor rhinitis, nonallergic rhinitis with eosinophil syndrome, and rhinitis medicamentosa are best treated with intranasal corticosteroids. Irrigation and debridement are the standard treatment of atrophic rhinitis, with an occasional course of antibiotics if needed for acute infection. For gustatory rhinitis, pretreatment with ipratropium bromide can be used.

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INTRODUCTION

Rhinitis is defined as inflammation of the mucous membrane lining in the nasal passages.[1–4] Rhinitis presents as nasal congestion, sneezing, nasal and palatal itching, rhinorrhea, and postnasal drainage. The categories of rhinitis include allergic, nonallergic, and infectious. A diagnosis of rhinitis should lead the provider to consider other coexisting conditions, such as asthma.

EPIDEMIOLOGY

Rhinitis is viewed by some as a trivial disease, but it may impair work, school, daily functioning, sleeping patterns, and quality of life.[4] Fifty-eight million people have allergic rhinitis, and 19 million people have nonallergic rhinitis, in United States.[2] Twenty-six million Americans have mixed rhinitis. Mixed rhinitis has components of both allergic and nonallergic rhinitis.[2] In 2002, the estimated direct and indirect costs to society for rhinitis were nearly US$11.6 billion dollars.[4]

Seventy percent of patients with allergic disease acquire the disease in childhood, and 70% of patients with nonallergic disease acquire it in adulthood, at more than 20 years of age.[2] Nonallergic rhinitis has a female predominance.

ALLERGIC RHINITIS

Classification

No standard classification exists for rhinitis. Allergic rhinitis is classified in the literature according to its seasonality, its perennial nature, or its occupational association. Seasonal allergic rhinitis is commonly caused by a variety of pollen allergens. Perennial allergic rhinitis is mainly caused by dust mites and animal dander. Occupational rhinitis results from airborne particles in the workplace and is a response to allergic and nonallergic mechanisms. Examples of occupational allergic antigens include animals and wood dust exposure. Chemicals and other irritants are examples of occupational nonallergic antigens. Infectious rhinitis is caused by infectious agents such as rhinovirus and may be complicated by bacterial infection.

Rhinitis can also be classified by whether it is persistent, intermittent, or episodic. Persistent rhinitis lasts longer than 4 weeks or more than 4 days per week. Intermittent rhinitis is defined as symptoms that last less than 4 weeks and fewer than 4 days per week. Episodic rhinitis may occur with sporadic inhalant aeroallergen exposure not typically encountered by the patient's usual indoor and outdoor environments, such as visiting a house with pets. This article uses the seasonal, perennial, and occupational rhinitis descriptors.

Regardless of the classification terminology used, all forms of rhinitis are further characterized by their severity. Mild rhinitis is described as rhinitis that does not impair work, school, daily functioning, or sleeping patterns. Moderate to severe rhinitis interferes with quality of life and disturbs activities of daily living and/or sleep. Severe rhinitis is so marked that normal functioning cannot take place without treatment.

Pathophysiology

Allergic rhinitis is the result of an immunoglobulin (Ig) E-mediated allergic reaction, with varying degrees of nasal inflammation.[1] Allergic rhinitis results from a type I hypersensitivity response to an inhaled allergen.[5] Allergens are proteins derived from airborne particulate matter, including dust mite feces, pollens, animal dander, and cockroach particles.[5]

Antigen-presenting cells (APCs) engulf allergens in the nasal mucosa.[5] The cells break the allergens into antigenic peptides.[5] APCs present the antigenic peptides to naive T cells (Th0).[5] When activated, Th0 cells differentiate into the Th2 subtype.[5] The Th2 cells release interleukin (IL)-4, which stimulates B cells to produce IgE.[5] IgE attaches to mast cells and basophils and renders them sensitized to the allergen.[5] When sensitized, mast cells and basophil cells are exposed to the allergen, and they degranulate.[5] Degranulation of these cells releases a host of mediators including histamine and prostaglandin.[5] Mast cell release of histamine is a major pathway for nasal inflammation in seasonal and perennial allergic disease.[1] Leukotrienes, kinins, and prostaglandins also play a role in the allergic pathway.[1]

Early and late phase reactions

Allergen exposure produces symptoms within minutes of the exposure and diminishes by 1 hour. Allergic reactions consist of early phase and late phase reactions.[5] Early phase reactions involve the mediation of IgE-allergen complexes and produce the typical symptoms of allergic rhinitis.[5] Late phase reactions involve activation and sequestration of inflammatory cells that produce vascular congestion and nasal hyperresponsiveness.[5] Inflammatory cells that migrate into the nasal mucosa include eosinophils, basophils, monocytes, T cells, and neutrophils.[5] The resultant inflammation causes the normal nasal response to become exaggerated.[5] Because of the nasal hyperresponsiveness, some individuals with allergic rhinitis can experience allergic symptoms on exposure to nonallergic stimuli (such as perfumes, smoke, strong odors, or other irritants).[5]

Eosinophils play a major role in nasal hyperresponsiveness.[5] Eosinophils arrive after allergen exposure and produce IL-5. IL-5 promotes activation and survival of other eosinophils.[5] Eosinophils also release toxic products that damage local mucosal cells.[5]

Prevention

Identifying the environmental allergens or irritants that trigger rhinitis symptoms can be important in the management of the disease process.[4] Mild disease can usually be managed with avoidance measures alone.[1] Allergen removal can also improve the severity of allergic rhinitis and can reduce the need for medications.[1] Pollen, dust mites, animals, insect proteins, and fungi are the most prominent allergic triggers.[4] Environmental control may take weeks to months to produce full beneficial effects, and complete avoidance of allergen is usually not feasible or practical.[1]

Reduction of dust mite allergen exposure can be accomplished in the following ways: remove carpets and soft toys, use covers impermeable to allergens for mattresses and pillows, vacuum beds weekly, and wash bedding at 60°C (140°F).[1] Pet dander avoidance can only be effectively managed by removing the pet and carefully cleaning all carpets, furniture, and mattresses.[1]

Implementation of avoidance measures is largely based on consensus panel recommendations because of the lack of high-quality evidence.[6,7] As with any treatment regimen or avoidance strategy, subpopulations of patients may respond differently to the same treatment plan.[8] A patient's treatment plan requires individualization based on factors such as age, symptom severity, seasonality, route of medication administration preference (ie, nasal vs oral), medication side effects, cost, onset of action, and benefit to comorbid conditions.[8]

Avoidance is the first-line treatment of any form of rhinitis. The Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines: 2010 Revision recommended environmental control measures for allergic rhinitis prevention (**Box 1**).[6]

Box 1
Environmental control measures for the prevention and treatment of allergic rhinitis

- Dust mite control
  - Encase bedding in dust mite covers
  - Wash bedding and toys at 60°C or hotter
  - Avoid carpets
- Eliminate or reduce occupational exposure to allergens
- Use small particulate furnace filters
- Pollen control
  - Keep windows closed
  - Use air conditioning
  - Limit outdoor exposure
- Mold control
  - Avoid basements
  - Reduce household humidity
- Pets
  - Avoid pets in the home
  - Confine pets to uncarpeted rooms
  - Bathe pets frequently
  - Equip rooms with high-efficiency particulate air filters

The ARIA guidelines make recommendations for children and pregnant or lactating women. Women should breast-feed for the first 3 months of life irrespective of atopic family history. The ARIA guidelines also state that pregnant and lactating women should not avoid food-related antigens for the prevention of childhood allergic rhinitis. In addition, children and pregnant women should completely avoid tobacco smoke exposure.[6]

Diagnosis

Allergic rhinitis is represented by sneezing, nasal congestion, nasal pruritus, and rhinorrhea.[2] When allergic disease is suspected, a skin prick test can be performed for confirmation.[1] When skin testing is difficult to interpret or not feasible (ie, dermatographism), allergen-specific IgE serum testing based on the patient's history can be useful.[1] The specific in vitro assays chosen should be based on local prevalence of aeroallergens.[4]

History

The history can be an important component in the differentiation of rhinitis. Nasal pruritus, palatal pruritus, sneezing, allergen exposure, seasonality, and associated ocular symptoms suggest allergic disease.[3,8] A family history of allergy is also an important clue that symptoms might be allergy related.[9]

Treatment

Oral antihistamines should be used to treat patients with mild or occasional seasonal allergic rhinitis. Cromolyn is an alternative to oral antihistamines in mild disease.[1]

Intranasal corticosteroids are effective in treating seasonal allergic rhinitis of moderate or long duration. If the patient's seasonal allergic rhinitis is severe, an oral antihistamine can be added to the intranasal corticosteroid. Other considerations for severe seasonal allergic rhinitis include adding oral decongestants, ipratropium bromide, or analgesics. As a last resort, immunotherapy should be considered for those who fail other therapies.[1]

Primary treatment of perennial allergic rhinitis is avoidance. Oral or intranasal antihistamines are effective for mild or intermittent symptoms not controlled with avoidance. Moderate or frequent symptoms should be treated with intranasal corticosteroids. Adding an oral antihistamine can be done if corticosteroids alone are not effective.[1]

Severe symptoms usually require additional therapies such as topical or oral decongestants for congestion and ipratropium bromide for rhinorrhea. Immunotherapy can be considered, but is not as effective in treating perennial allergic rhinitis as it is in treating seasonal allergic rhinitis.[1] As a last resort, surgical reduction of turbinate hypertrophy is an option.

Therapies not recommended by the ARIA guidelines include acupuncture, butterbur, herbal medicines, phototherapy, or other physical techniques.

NONALLERGIC RHINITIS

Classification

Nonallergic rhinitis is subdivided into several categories (**Box 2**). Nonallergic rhinitis is also known as perennial nonallergic rhinitis, idiopathic rhinitis, and vasomotor rhinitis.[3]

Box 2
Nonallergic rhinitis classification

- Vasomotor: associated with irritants and change in temperature and humidity, alcohol, exercise; involves activation of neural efferent pathway to nasal mucosa, anticholinergic mediated; may be autonomic dysfunction.
- Gustatory rhinitis: nasal congestion associated with ingestion of foods, alcoholic beverages.
- Nonallergic rhinitis with eosinophil syndrome (NARES): paroxysms of symptom flares including sneezing, watery rhinorrhea, nasal itching, congestion, and some anosmia; nasal eosinophils are present, but systemic allergy is lacking.
- Rhinitis medicamentosa: caused by prolonged and repetitive use of topical nasal decongestants. Also associated with cocaine use. Patients have rebound congestion.
- Occupational rhinitis: symptoms from irritating chemicals, grain dust, laboratory animal antigens, wood, ozone. May coexist with occupational asthma.
- Hormonal rhinitis: rhinitis related to pregnancy or the menstrual cycle. Symptoms resolve 2 weeks after delivery. Sinusitis is 6 times more common in pregnancy.
- Drug-induced rhinitis: associated with angiotensin-converting enzyme (ACE) inhibitors, phosphodiesterase-5 selective inhibitors, alpha receptor antagonists, phentolamine.
- Atrophic rhinitis: caused by glandular cell atrophy. Symptoms include nasal crusting, dryness, and fetor. Abnormally wide nasal cavities; squamous metaplasia of nasal mucosa.
- Cold air–induced rhinitis: symptoms occur on exposure to cold air.
- Anatomic rhinitis: caused by polyps, tumors, septal disturbances.

Data from Wallace D, Dykewicz M. The diagnosis and management of rhinitis: an updated practice parameter. J Allergy Clin Immunol 2008;122:S1–84.

Pathophysiology

A normal nasal response is how the nasal mucosa normally functions in response to exogenous physical stimuli. Hyperresponsiveness is the exaggeration of the normal nasal mucosa response to any stimuli. Genetic or pathologic factors can alter how the nasal mucosa functions in response to these stimuli.[5]

The pathophysiology of nonallergic rhinitis does not involve IgE mediation. The specific mechanism is poorly understood because of the many presentations of the conditions in this category.[2,3] What is known about the pathophysiology is that the nonallergic rhinitis is caused by nasal hyperactivity to nonimmunologic stimuli, but little knowledge exists regarding the exact mechanism of this nasal hyperactivity.[2] Autonomic dysregulation and nociceptive nerve dysfunction are proposed mechanisms of nasal hyperresponsiveness.[3] Because of its lack of an allergic component, nonallergic rhinitis is frequently distinguished from allergic rhinitis by negative IgE testing and/or negative skin prick testing.[2]

Diagnosis/History

The diagnosis of nonallergic rhinitis is driven by the patient's history of symptoms (see **Box 2**). Specific diagnostic tests may help clarify the diagnosis, although they are often unnecessary.

Treatment

Because of the variance in causes of nonallergic rhinitis, treatments also vary. **Box 3** summarizes specific treatments. Vasomotor rhinitis, NARES, and rhinitis medicamentosa are best treated with intranasal corticosteroids. Irrigation and debridement are the standard treatment of atrophic rhinitis, with an occasional course of antibiotics if needed for acute infection. For gustatory rhinitis, pretreatment with ipratropium bromide can be used.

LOCAL ALLERGIC RHINITIS

Local allergic rhinitis is a newly recognized subset of rhinitis. The allergic reaction of local allergic rhinitis is confined to the nose. The local inflammatory response is similar to allergic rhinitis, but a systemic response is lacking. Some patients previously classified as having nonallergic rhinitis are now being found to have local allergic rhinitis.[10] Local allergic rhinitis is confined to the nasal mucosa and is characterized by local inflammatory reactions including local infiltration of eosinophils and localized detection of IgE in response to aeroallergens.[10] Patients with local allergic rhinitis do not have

Box 3
Treatment of nonallergic rhinitis

1. Vasomotor rhinitis: intranasal corticosteroids
2. NARES: intranasal corticosteroids
3. Atrophic: irrigation, debridement, antibiotics as needed
4. Gustatory: ipratropium bromide
5. Occupational: avoidance
6. Hormonal: nasal saline
7. Rhinitis medicamentosa: intranasal corticosteroids or oral steroids

significant systemic reactions to aeroallergens including negative skin prick testing and negative serum IgE levels.[10] Local allergic rhinitis is associated with asthma and conjunctivitis and commonly begins in childhood.[10] The prevalence of local allergic rhinitis is unknown and its clinical relevance is unclear.[10] However, in a small study, the prevalence of local allergic rhinitis was approximately 26%.[10]

DIAGNOSTIC TESTING

Testing that can be performed in the evaluation of allergic or nonallergic rhinitis includes skin prick testing, which can be helpful to rule in or rule out allergic disease.[4] Total serum IgE or IgG levels should not be routinely performed. Total serum IgE levels have low sensitivity for predicting allergic disease. IgE testing, specific to local aeroallergens, may be indicated if skin prick testing is not feasible or is difficult to interpret, such as in a patient with dermatographism. Increased serum IgG levels have been suggested as a risk factor for atopy, but evidence has been inconclusive.[4] Nasal smears to assess for eosinophils are not recommended for routine use.[8]

Fiberoptic nasal endoscopy can be helpful, but is expensive and should be reserved for those patients with atypical symptoms or those with inadequate response to treatment.[4]

Rhinomanometry measures the airflow obstruction in the upper airway and can give the clinician an objective measurement of nasal congestion in response to certain interventions. Rhinomanometry can be useful in assessing the severity of an anatomic abnormality and assessing those patients with obstructive sleep apnea.[4] A sleep study should be considered in patients presenting with chronic rhinitis, because chronic rhinitis is a risk factor for sleep disordered breathing.[4]

Computed tomography scans and magnetic resonance imaging are expensive but can be used to identify anatomic abnormalities using coronal sections. However, this type of imaging may not correlate well with functional obstruction.[4]

Patients with watery rhinorrhea and recent nasal surgery or trauma may have a cerebrospinal fluid (CSF) leak. Beta transferrin protein testing can be performed on the rhinorrhea and can confirm a CSF leak.[4]

DIFFERENTIAL DIAGNOSIS

Diagnoses to consider when a patient presents with rhinitis are listed in **Box 4**.

Smelling difficulty may suggest nasal polyps and unilateral symptoms may signify an anatomic problem.[4,8] **Box 5** outlines questions that help in the diagnosis and classification of rhinitis and **Box 6** lists physical examination systems that should be evaluated in patients with rhinitis. Particular attention should be placed on examination of the nasal passages.

MEDICATIONS

Intranasal Corticosteroids

Intranasal corticosteroids (INCS) should be first-line treatment of allergic rhinitis in the moderate to severe category, and they are the most effective treatment of all nasal allergic rhinitis symptoms.[1,8,9] INCS are recommended in chronic rhinosinusitis.[9] There is no relevant difference between INCS preparations and no INCS preparation is more efficacious than another.[3] Budesonide is US Food and Drug Administration (FDA) indicated for the treatment of nonallergic rhinitis. The INCS mometasone has an indication for nasal polyposis.[9]

Box 4
Differential diagnosis of patients presenting with rhinitis

- Allergic rhinitis
- Nonallergic rhinitis
- Occupational rhinitis
- Structural/anatomic factors
  - Deviated septum
  - Nasal and sinus tumors
  - Nasal turbinate hypertrophy
  - Nasal polyposis
- CSF rhinorrhea
- Pharyngonasal reflux
- Infectious rhinitis
  - Rhinovirus
  - Coronavirus
  - Bacterial infection
- Chronic sinusitis
- Ciliary dyskinesia syndrome
- Systemic disorders
  - Wegener disease
  - Tuberculosis
  - Syphilis
- Medication side effects
  - ACE inhibitors
  - Phosphodiesterase-5 selective inhibitors
  - Alpha receptor antagonists
  - Phentolamine
- Hormonal disturbances
- Aspirin intolerance
- Exogenous noxious stimuli
- Rhinitis sicca (chronically dry mucous membranes)
- Atrophic rhinitis
- Local allergic rhinitis
- Rhinitis medicamentosa
- Vasomotor rhinitis
- Gustatory rhinitis
- NARES
- Cold air–induced rhinitis

Data from van Cauwenberge P, Bachert C, Passalacqua G, et al. Consensus statement on the treatment of allergic rhinitis. European Academy of Allergology and Clinical Immunology. Allergy 2000;55:116–34; and Wallace D, Dykewicz M. The diagnosis and management of rhinitis: an updated practice parameter. J Allergy Clin Immunol 2008;122:S1–84.

Box 5
Components of the history important to consider when evaluating the patient with rhinitis

- Pattern (perennial, seasonal, or combination)
- Triggers
- Family history
- Current medications
- Previous response to treatment
- Comorbid conditions
- Environmental history
- Occupational exposures

Data from Wallace D, Dykewicz M. The diagnosis and management of rhinitis: an updated practice parameter. J Allergy Clin Immunol 2008;122:S1–84.

Side effects of INCS include nasal burning, irritation, stinging, epistaxis, and local dryness.[3] Mucosal atrophy is not a noted side effect. Local candidiasis is rarely seen with INCS use.[3] INCS have negligible hypothalamic-pituitary-adrenal axis suppression and the systemic burden of intranasal corticosteroids is clinically insignificant.[3]

The mechanism of action of INCS involves the reduction of the inflammatory response. Glucocorticoids move through the outer cellular membrane, binding to intracellular receptors.[3] Intracellular steroid complexes are transported to the cell nucleus where responsive genes are either upregulated or downregulated.[3] Gene modulation leads to a reduction in proinflammatory mediators in the mucosa.[3]

Oral Antihistamines

Oral antihistamines reduce nasal itching, rhinorrhea, and sneezing but do not treat nasal congestion effectively.[1] Oral antihistamines can reduce conjunctivitis and urticaria.[1]

First-generation oral antihistamines are poorly selective for H-1 receptors and can cause anticholinergic effects by blocking muscarinic receptors. Blockade of the muscarinic receptors causes dry mucous membranes, blurry vision, constipation, tachycardia, and urinary retention.[3] Because first-generation antihistamines cross the blood-brain barrier, significant sedation occurs.[3] The sedative effects of these antihistamines have been linked to industrial accidents and contribute to significant loss of function at work and school.[3] Overall, first-generation antihistamines are limited in usefulness because of these sedative and anticholinergic effects.[3]

Second-generation antihistamines have better selectivity for the H-1 receptor.[3] A more complex molecular structure allows second-generation antihistamines to not cross the blood-brain barrier. These antihistamines are considered nonsedating.[9] The risk/benefit ratio of second-generation antihistamines is so favorable that they are considered first-line therapy for mild allergic rhinitis. Tolerance to antihistamines has not been shown to occur.

Intranasal Antihistamines

Azelastine 0.1% has an indication for allergic and vasomotor rhinitis and is the only nasal antihistamine available in the United States.[3] Intranasal antihistamine (INA)

Box 6
Systemic physical examination of patient with rhinitis

- General
  - Elongated facies
  - Mouth breathing
- Ears
  - Tympanic membrane
    - Air fluid level
    - Erythema
    - Retraction
    - Mobility
- Nose
  - External crease
- Nasal mucosa[2]
  - Bluish discoloration
  - Red nasal mucosa
  - Excessive watery-clear mucus
  - Polyps
  - Chronic sinusitis
  - Turbinate hypertrophy
  - Crusting
  - Discharge
  - Septal deviation
  - Septal perforation
- Eyelids
  - Venous stasis of the lower lids (allergic shiners)
- Conjunctiva
  - Edema
  - Erythema
  - Excessive lacrimation
  - Cobblestoning
- Oropharynx
  - High-arch palate
  - Tonsillar enlargement
  - Adenoid enlargement
  - Cobblestoning
- Neck
  - Thyroid examination
  - Lymphadenopathy

- Respiratory
  - Wheezing
- Abdomen
  - Liver enlargement
  - Spleen enlargement
- Skin
  - Eczema
  - Urticaria
  - Dermatographism

Data from Wallace D, Dykewicz M. The diagnosis and management of rhinitis: an updated practice parameter. J Allergy Clin Immunol 2008;122:S1–84.

may be better than oral antihistamines in reducing total nasal symptom scores.[3] Nasal antihistamines can help control nasal congestion.[9]

Side effects of intranasal antihistamines include bitter taste, headache, epistaxis, and nasal irritation.[3,9] Nasal antihistamines do not produce a significant amount of sedation.[1] INA has the advantage of a quicker onset of action for nasal and ocular symptoms compared with INCS.[1]

Nasal antihistamines need to be administered twice daily for maximum clinical benefit.[1]

Intranasal Ipratropium

Intranasal ipratropium (IP) controls seromucous gland secretion by blocking muscarinic receptors, so it is effective in treating watery rhinorrhea.[1] IP is useful in treating nonallergic rhinitis and the common cold.[1] Ipratropium does not improve nasal congestion or sneezing.[1] Side effects of IP include nasal dryness, burning, irritation, stuffy nose, headache, and dry mouth.[1]

Because of its onset of action, nasal anticholinergics (ipratropium) as well as nasal corticosteroids, nasal antihistamines, and oral antihistamines, are effective in the treatment of episodic allergic rhinitis.[8]

Leukotriene Receptor Antagonists

Montelukast is the only leukotriene receptor antagonist (LTRA) that is FDA approved for allergic rhinitis treatment, but it has a limited role in nonallergic rhinitis.[3] LTRAs should be used as second-line or third-line therapy[3] and should not be used in those patients with episodic symptoms.[8]

Nasal Cromolyn

Nasal cromolyn can be used before allergen exposure for prophylaxis of episodic allergic rhinitis.[9] Cromolyn should be considered for early mild rhinitis, but should not be considered a first-line treatment in allergic rhinitis.[1] Nasal cromolyn is mostly void of side effects. It should be administered 3 to 4 times a day.[1,9] The mechanism of action is poorly understood, but evidence suggests that it inhibits mast cell degranulation.[9]

Decongestants

Oral and topical decongestants may be used for symptomatic relief in patients with nonallergic rhinitis.[3] Oral and topical decongestants affect the alpha adrenoreceptors

on nasal capacitance blood vessels.[3] Nasal capacitance blood vessels are responsible for mucosal swelling resulting in nasal congestion.[3] Oral decongestants act on the sympathetic tone of blood vessels through the adrenergic pathway causing vasoconstriction.[1]

Topical phenylephrine and topical oxymetazoline are over-the-counter topical decongestants.[9] Topical decongestants are effective in treatment of nasal congestion, but should not be used for more than 5 days because of the risk of rebound vasodilatation, and subsequent rebound congestion (rhinitis medicamentosa).[1,9] Tolerance may be prevented by concurrent use of INCS.[3]

Side effects of topical decongestants are stinging, burning, dryness, and mucosal ulceration (less common).[3] Oral decongestant side effects include insomnia, anxiety and tremors, tachycardia, increases in blood pressure, and palpitations.[3] Oral decongestants should be not be used in young children and adults more than 60 years of age.[1]

Combination Therapies

Combination therapy may provide a therapeutic advantage, but cost must be considered and it may reduce compliance.[8] Fluticasone with azelastine is superior to INCS alone for moderate-severe allergic rhinitis.[3,8,9] Oral antihistamines with oral decongestants are more effective than antihistamines alone for nasal congestion relief.[8]

Intranasal anticholinergics and intranasal corticosteroid combination therapy is more effective for relief of rhinorrhea than either drug as monotherapy.[8] Although inferior to intranasal corticosteroid treatment, an oral antihistamine with montelukast has an additive effect for the treatment of allergic rhinitis.[3]

Nasal Saline

Nasal saline plays a role in allergic and nonallergic rhinitis by promoting ciliary clearance, and removing mucus and other irritant materials.[3] Some evidence suggests that hypertonic saline provides modest benefit compared with isotonic saline.[3]

Anti-IgE

Omalizumab is a monoclonal antibody available for the treatment of poorly controlled asthma, but it might have a role in the treatment of allergic rhinitis.[3] Omalizumab has proven efficacy in allergic rhinitis, but is limited because of high cost, anaphylaxis reports, and availability in only injectable form.[3]

Omalizumab binds to IgE and hinders its relationship with inflammatory cells.[3] Omalizumab only binds to circulating IgE and not bound IgE.[3]

Systemic Steroids

Systemic steroids should not be used as first-line treatment of allergic rhinitis. The risk of adverse effects of systemic steroids precludes using them for longer than 3 weeks. Systemic steroids should not be used in children or pregnant women. Because few studies are available to support the use of systemic steroids, they are used as a last resort.[1]

Immunotherapy

Subcutaneous immunotherapy

Subcutaneous immunotherapy (SCIT) involves the subcutaneous injection of aqueous extracts of an offending allergen. By starting at low doses of the injected allergen and by progressively increasing the dose, SCIT builds up immunity to the offending allergen.[11] SCIT is indicated for adults and children presenting with pollen-induced

and dust mite–induced allergic rhinitis, and in those patients for whom medications and avoidance measures are inadequate.[11] SCIT is the only treatment that has proved to alter the course of allergic disease.[9] Evidence suggests that immunotherapy can halt the advancement of allergic disease that causes asthma and that it halts the creation of new sensitivities.[11] Children, patients early in the disease process, and patients with few sensitivities are the most likely to benefit from SCIT.[11]

SCIT starts with a buildup phase, which entails increasing the injected allergen dose over time. When the maximum dose of allergen extract is reached, the maintenance phase begins.[1] The maximum dose of allergen in the injections is variable among the different allergen extracts. For example, the maximum dose of dust mite allergen is 7 μg.[11] The usual recommendation is that maintenance treatment should last for 3 years.[11] After an average of 3 to 4 years of maintenance immunotherapy, the effectiveness of treatment lasts for 3 years or more after discontinuation.[11]

The inherent risk of SCIT is systemic anaphylaxis.[1,11] The rate of significant systemic reactions in patients with allergic rhinitis is approximately 5%.[1] Times of the year with increased pollen count can predispose to severe and life-threatening systemic reactions to SCIT.[11] Patients must wait 20 to 30 minutes after SCIT treatment to monitor for severe reactions.[11] Pretreatment with antihistamines has the potential to reduce systemic reactions.[11]

MONITORING

The Total Nasal Symptoms Score is a subjective assessment of a patients' specific symptoms, including rhinorrhea, nasal congestion, sneezing, and pruritus, and can be used to assess effectiveness of medications.[8] The Rhinoconjunctivitis Quality of Life Questionnaire is another tool that can be used for monitoring of therapy.[8] Based on the results of the screening tool used, the provider should consider stepping down treatment when the patients' symptoms have been controlled.[8]

Consultation with an allergist is based on several factors. Patients under evaluation for allergy testing and immunotherapy should be considered for consultation with an allergist/immunologist. Other instances in which a consultation might be considered is if a patient has a severe and prolonged course of rhinitis; if there are comorbid conditions present, such as asthma, chronic sinusitis, or nasal polyps; or if symptoms are interfering with quality of life or ability to function.[4]

INFECTIOUS RHINITIS/RHINOSINUSITIS

Infectious rhinitis and rhinosinusitis are synonymous with sinusitis. Acute rhinosinusitis most commonly occurs as a complication of viral upper respiratory infections.[9] Viral upper respiratory infections cause mucosal edema leading to obstruction of the sinus openings as well as ciliary function impairment.[9] The low-oxygen environment and the stagnant mucus of the sinuses allow viral and bacterial proliferation.[9]

Chronic rhinosinusitis is the result of long-term obstruction and/or function of the sinuses.[9] Chronic inflammation of the nasal mucosa leads to chronic low-grade infections.[9]

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