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REVIEW ARTICLE

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Management Of Allergic Rhinitis

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ABSTRACT

Allergic rhinitis (AR) is an IgE mediated hypersensitivity of the mucous membrane of the nasal airways characterized by nasal symptoms, such as nasal congestion, rhinorrhoea , sneezing and itchy nose.The management of AR consists of allergen avoidance, when possible and the oral and intranasal H_1antihistamines,intra-nasal corticosteroids, leukotriene modifiers, mast cell stabilizers and decongestants .

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KEYWORDS

allergic rhinitis ,corticosteroids, anti-histamines, leukotriene antagonists

INTRODUCTION

Allergic rhinitis (AR) is an IgE mediated hypersensitivity of the mucous membrane of the nasal airways characterized by nasal symptoms, such as nasal congestion, rhinorrhoea, sneezing and itchy nose [1,2]. It affects a large percentage of pediatric patients and causes significant number of school days missed per year. Impairment of work in adults also occurs affecting the finances of patients indirectly through lost workdays and directly through healthcare cost spent for the disease[3].

AR is divided into seasonal AR (SAR) and perennial AR(PAR). SAR symptoms occur during a specific season in which aeroallergens such as tree and grass pollen in the spring and summer and weed pollens in the autumn are present in out door air. It presents with more rhinorrhoea , pruritus and sneezing. PAR symptoms are present throughout the year and are triggered by dust mite, animal dander, indoor molds and cockroaches. Sneezing, itching and nasal discharge are prominent but rhinorrhoea may be more viscous or purulent[1].

Pathogenesis

AR develops due to the activation of mast cells upon exposure to an irritant .Mast cells degranulate, releasing various enzymes and inflammatory mediators, including histamine, prostaglandin D and leukotrienes (LTc_4, LTD_4, LTE_4). Inflammatory mediators increase the permeability of membrane and there is leakage of fluids which stimulates nerves and causes itching and sneezing. There is infiltration of inflammatory leukocytes, regulated by cytokines, chemokines and adhesion molecules. Inflammatory responses are restimulated leading to further leakage of fluid and congestion.[1]

Diagnosis of Allergic Rhinitis

Clinical history is essential for an accurate diagnosis of AR, assessment of severity and response to the treatment. Examination of the nose, done with a nasal speculum, reveals that the nasal mucosa appears pale and swollen, with a bluish-grey appearance in severe mucosal edema. Mucosa is red in acute infections and with over use of topical medications.

Accentuated folds below the margin lines of the inferior eye lid, i.e. Dennie-Morgan lines might be present . Infraorbital dark skin discoloration can be present with nasal obstruction Ear should be examined for evidence of associated otitis media[4].

Several studies suggest an association between AR and asthma[2] and other morbidities like sinusitis, sleep impairment, fatigue and learning speech impairment. So patient should be evaluated to rule out the above problem**s**[5].

Diagnosis confirmation of AR after taking history and doing physical examination can be done by skin testing or radio-allergosorbent testing (RAST). Skin test is the fastest, cheapest and most accurate way of testing. It involves introduction of allergen extract into the skin by prick or intra- dermally. The wheal and flare response to a specific allergen is then compared with the control. In-vitro measurement of allergen specific IgE can be done using RAST test [4].

Management of allergic rhinitis

The management of AR consists of allergen avoidance, when possible and the oral and intranasal H_1 antihistamines, intranasal corticosteroids, leukotriene modifiers, mast cell stabilizers and decongestants. In selected patients allergen specific immunotherapy is used that is disease modifying[6,7].

1. ***Allergen Avoidance***
   It is very difficult and seems impractical. However, regular medication use can be avoided by controlling exposure to indoor allergens by adopting some of the measures, that includes using of a bleaching agent on tiles, sinks, shower walls and avoiding humidifiers. Low pile type of carpets should be preferred. Pillows and mattresses should be put in airtight plastic encasing. Furry pets should not be kept. Keep the windows closed to avoid outdoor pollens and use air conditioning with external vents closed. Wear masks while cleaning the room or mowing grass.[6]

2. ***Anti-allergic drugs***
   — Those for prophylaxis e.g. steroids, sodium cromoglycate, ketotifen.

   — those controlling acute symptoms e.g. antihistamines, decongestants, Leukotriene receptor antagonists.

(a) Intranasal Corticosteroids

Intranasal corticosteroids are the first time of therapy for moderate-severe AR. The available once daily intranasal steroids are triamcinolone acetonide, budesonide, fluticasone and mometasone. They affect the inflammatory mechanisms of the early and late phase allergic processes and are effective in controlling symptoms of AR. Adverse effects may be delivery system related as freon delivered aerosols may lead to bleeding, drying and crusting of the nasal mucosa. Children should get the lowest possible dose of intranasal corticosteroid and they should have routine height monitoring to look for suppression of linear growth[1].

The systemic side effects of the corticosteroids are not much of a problem as the delivery is only local. However, some studies have suggested a link among posterior subcapsular cataracts, glaucoma and intranasal corticosteroids[1,2].

(b) Sodium Cromoglycate

It inhibits the degranulation of the mast cells and prevents the release of histamine and other mediators of the allergic response. It has to be given 4 times daily and is used for prophylaxis[6].

(c) Ketotifen

It has both mast cell stabilizing and antihistaminic activity. It can also be used orally in a dose of 1-2 mg twice daily for prevention of allergic rhinitis. It can lead to sedation and Weight gain.[6]

(d) Anti-histamines

They are the most effective drugs for the relief of acute symptoms. They act by blocking H, receptors. The older first generation H, antihistamines such as diphenhydramine, chlorpheniramine and promethazine are sedating antihistamines as they cross the blood-brain barriers, leading to impaired performance at home, work and school. Apart from their antihistaminic activity, they also cause anticholinergic and antiserotonergic activity.

The newer second generation oral H, antihistamines like cetrizine , fexofenadine, loratidine and topical azelastine are largely free from anticholinergic sedative effects of the classical antihistamines and have a longer duration of action. Both oral and

topical newer antihistamines are recommended as the first line therapy for the treatment of mild to moderate AR[8].

(e) Decongestants

Decongestants reduce nasal congestion by activating α-adrenergic receptors on the nasal vessels leading to vasoconstriction. A combination of pseudoephedrine and an antihistamine has been found to be significantly more effective in reducing total nasal symptoms than either agent alone.

(f) Leukotriene reactor antagonists

Leukotrienes are important mediators of nasal allergic reactions involved in both early and the late-phase allergic response. Studies have shown that montelukast is as effective **as effective** as antihistaminic and using both fexofenadine and montelukast showed significantly better control of nasal congestion, showing that leukotriene receptor antagonist- antihistamine combination is more effective than antihistamine alone in the control of allergic rhinitis symptoms[9,10].

(g) Immunotherapy

Subcutaneous immunotherapy (SCIT) is indicated for the treatment of AR, in patients who continue to have moderate-severe symptoms despite antiallergic therapy or are not able to tolerate pharmacotherapy or have co-existing asthma. It is the only therapy that can alter the natural course of the disease.[1]

SCIT consists of serial injections of allergen extracts, till the maintenance dose has been achieved or the maximal tolerated dose is reached, to provide protection from natural exposure to antigens which induce the symptoms of AR.

The objective efficacy of hypersensitizing preparations have only been established in perennial rhinitis and asthma due to house dust mite, seasonal allergic rhinitis and anaphylaxis due to bee or wasp stings.[11]

Drawbacks to SCIT include the frequent injection schedule and the requirement for the injection to be done in the hospital with a two hours observation period. The most common adverse effects associated with SCIT are local injection site swelling and erythema. Systemic reaction including anaphylaxis may occur. Immunotherapy should be given under close medical supervision with full facilities for cardio-pulmonary resuscitation. An antihistaminic may be given 30 minutes prior to each injection. Adrenaline should always be readily available[12].

(h) Other modalities

These mainly include sublingual immunotherapy and endonasal phototherapy.

Sublingual immunotherapy (SLIT)

A study showed that SLIT imporved the symptoms in Korean patient with AR from hence dust mites. Laboratory parameters including eosinophil counts and specific IgE were modified after 1 year SLIT.[13]

Another study provides evidence that quality of life can be improved in polysensitized patients treated with SLIT. The use of one or two allergen extracts seems to be sufficient and effective in terms of improving the quality of life.[14]

Endonasal Phototherapy

The literature documents the fact that UV irradiation of cutaneous langerhans cells in vivo prevents the development of contact allergy and produces long lasting immunosuppression. Endonasal phototherapy

combination of UVB (5%), UVA (25%) and visible light (70%) utilizes the immunosuppressive effects of UV irradiation. All patients exposed to above therapy showed a significant clinical benefit post- treatment as assessed by standardized instruments including total nasal symptom score and nasal congestion score etc.[15,16]

CONCLUSION

AR affects the quality of life of the patient significantly and it poses a challenge before the physician to manage it with drugs that are efficacious as well as safe. Second generation antihistamines and leukotriene receptor antagonists seem to be fulfilling the need to some extent. Still preference of one over the other and safer use in children needs to be established fully.

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