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Allergic Rhinitis (M Calderon, Section Editor)

Treatment of Allergic Rhinitis in Special Conditions

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Opinion statement

Allergic rhinitis is a global health problem with considerable socio-economic and personal health burden. Treatment of allergic rhinitis needs careful evaluation of comorbid conditions whilst ensuring sound symptom control. Wariness of perceived side effects, incomplete assessment and underestimation of disease impact, may all lead to under-treatment and needless suffering on part of the patient. This article examines the evidence base for treating allergic rhinitis in the most commonly encountered special conditions and makes recommendation for treatment based on up-to-date literature evidence.

Introduction

Allergic rhinitis (AR) is a global health problem with a heavy personal and social burden. Incidence and prevalence has been rising consistently, particularly in countries previously not burdened with this disease [1]. Lack of adequate recognition of symptoms and insight into optimal treatment limit the potential benefits to be gained from adequate therapy. Patients may be wary of 'overmedicating' particularly whilst using topical corticosteroids and the primary care setting may not always provide adequate workup, education and monitoring for this all too common condition. This chapter summarises the up-to-date evidence for the treatment of allergic rhinitis in special conditions.

Paediatric population

Half of all paediatric consultations are for ENT-related problems with nasal symptoms forming a large part of these consultations. Under the age of 14, prevalence of AR is greater in males with family history of atopy, than females. Familial history of allergic rhinitis is itself a particular risk factor for development of AR in children [2]. In a study of over a thousand children, allergic rhinitis and

exposure to cigarette smoke are risk factors for adenoidal hypertrophy with house dust mite the most common sensitivity [3]. Adenoidal hypertrophy in turn is associated with sleep-disordered breathing and chronic serous otitis media [4] demonstrating the close symptomatic symbiosis of these conditions. Ascertaining the presence of allergen specific IgE consistent with history of sensitisation is indicated for accurate diagnosis of allergic rhinitis. Skin prick testing is quick and cost effective with serum IgE indicated in cases of dermatographism, inability to wean of antihistamine treatment or non-compliance with skin prick testing particularly in the under 5 s [5••].

Antihistamines are highly effective in paediatric allergy [6] with regular administration better than 'as required' for control of inflammation. Highly lipophilic first generation antihistamines have greater central nervous system cognitive impairment with sedation a particular concern. Hepatic p450 metabolised antihistamines may be broken down to active or inactive metabolites making their plasma levels less predictable and may be influenced by other drugs with similar metabolisation pathways. Antihistamines will have rapid onset of action, and are metabolised quicker in children inferring twice daily administration (versus once a day use in adults). Compliance should be checked at 1 month, symptoms reassessed and treatment stepped up or down as appropriate (Fig. 1). Oral and intranasal antihistamines are both effective, with oral medication better tolerated but intranasal treatment possessing a faster onset of action. Meta-analysis results would recommend oral antihistamines over intranasal routes [7]. First generation antihistamines should not be used due to adverse safety profile. Of the second generation treatments, fexofenadine is likely to cause the least sedation [8]. Randomised controlled trial shows two or more weeks of nasal irrigation is an effective method of reducing antihistamine use [9•]. Cochrane review excluding studies that allowed rescue medication failed to demonstrate efficacy of intranasal

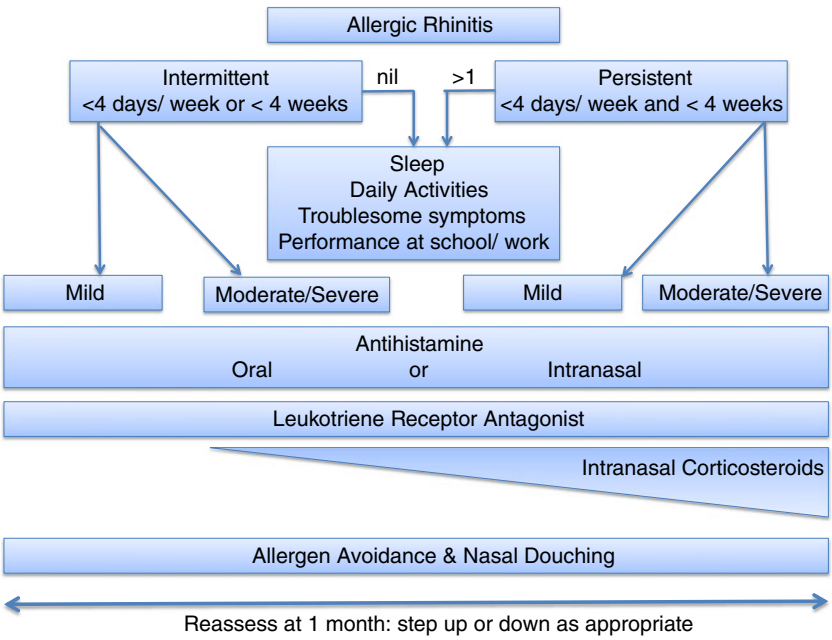

Fig. 1. Suggested treatment ladder for treatment of allergic rhinitis in paediatric population.

corticosteroids although there exists evidence of rapid onset of action and improvement of co-existent asthma with such treatment [10]. Mometasone, fluticasone proprionate and fluticasone furoate possess low bioavailability with no resultant impact on growth after a year use. Once daily administration will further make their use appropriate and effective in childhood allergic rhinitis.

There is no role for depot injections of systemic corticosteroids as they atrophy skin, muscle, reduce bone mineralisation and effect growth. Corticosteroids may exert growth restrictive effects by multiple mechanisms including decreased growth hormone release, augmented growth hormone receptor expression, decreased insulin-like growth factors 1 activity, altered collagen synthesis and suppression of adrenal androgen production. Despite this, when used within recommended dosages, restriction of growth is not a side effect of intranasal corticosteroid use in the paediatric population. Newer preparations have improved safety profile with better receptor binding and lower systemic bioavailability [11]. Specifically, mometasone furoate, budesonide, ciclesonide, fluticasone furoate and triamcinolone acetonide do not alter final adult height. Beclomethasone diproprionate use of over 1 year duration in perennial allergic rhinitis has shown evidence of reduction in standing height albeit without significant alteration in hypophyseal-pituitary axis measurements [12]. Oral corticosteroids may be used in exceptional circumstances with cases with co-existent allergic process such as asthma. Small paediatric and larger meta-analysis (including adults) demonstrate oral leukotriene antagonists as useful adjuncts to treatment particularly with co-existent asthma. Topical anticholinergics are rarely used in children and sodium chromoglycate, although effective, needs too frequent administration to achieve reliable compliance. A short course of nasal decongestants may be applicable with awareness of long-term use causing rhinitis medicamentosa. Overall, topical corticosteroids are better at controlling allergic rhinitis than either antihistamines or leukotriene receptor inhibitor with sodium chromoglycate less effective than all of the above [8].

Treatment of allergic rhinitis in childhood relies on accurate diagnosis with appreciation of concurrent comorbidities. Rhinitis of non-allergic, possibly infectious cause is common with possible co-existent symptoms of cough, fatigue, sleep-disordered breathing, eustachian tube dysfunction and reflux needing treatment independently whilst making diagnosis and treatment of allergic rhinitis more complex. Allergic rhinitis increases risk of asthma over threefold. Immunotherapy in monosensitised allergic rhinitic patients prevents development of new sensitivities and may prevent development of asthma [13]. Use of intranasal corticosteroids in AR improves lung function in children with impaired forced expiratory flow (FEF) and FEV_1 but is not able to match controls with AR and normal lung function [14].

A 2007 Cochrane review, using studies that include both adults and children, demonstrates subcutaneous immunotherapy (SCIT) to be effective in allergic rhinitis although due to method of administration, applicable to school-age children [15]. Sublingual immunotherapy (SLIT) is effective in treatment of paediatric allergic rhinitis, with longer than 18 months and pollen extract (versus house dust mite) demonstrating greater efficacy at reducing symptom scores and need for rescue medication [16]. Furthermore, SLIT is effective in pollen and house dust mite-driven allergic rhinitis albeit with considerable heterogeneity of studies [17•].

Treatment of allergic rhinitis in the paediatric population is summarised in

Fig. 1. Careful diagnosis, with particular attention to meticulous history, and documentation of potential comorbidity is the first step in the holistic approach to this all too common paediatric condition.

Elderly

Incidence of allergic rhinitis peaks in adulthood but a significant proportion of the over 65 s also suffers. Studies put the prevalence of allergic rhinitis in the elderly between 5.4 and 10.7 % with the above 50 age group reporting increased ocular symptoms [18]. Allergic rhinitis is prevalent in the elderly as demonstrated by the population based Swiss study, SAPALDIA, with up to 10 % of the over 65 affected. Particular considerations when considering pharmacotherapy are polypharmacy, comorbidity (including loss of memory), compliance and overall frailty. Ageing of the immune system, immunosenescence, will result in decline of IgE in non-atopic individuals, but this may not be reflected in sensitised allergics as demonstrated by skin prick responses, or specific RAST IgE measurements to inhaled or ingested allergens [19]. Both innate and adaptive immune responses decline in function. T and B cell repertoires are augmented with diminution of bone marrow and thymic output. Phagocytic, pinocytic, apoptotic an antigen presenting functions of granulocytes, leukocytes and dendritic cells are diminished, with the above effects coalescing into an overall impaired immune response [20].

Ageing of the nose with changes in septal and skin architecture may alter collumelar support with loss of collagen integrity and alterations of the extracellular matrix. Reduction of blood flow will effect the humidifying properties of the nose with both of the above factors potentially complicating presentation and management of allergic rhinitis in the elderly [21]. Age-related decrease in mucociliary function may worsen symptoms of allergic rhinitis [22]. With ageing skin, immune system and nasal mucosa, other types of rhinitis such as vasomotor, atrophic or non-allergic-prevalent in this age groups should be considered and treated if necessary [23].

First generation antihistamines should be used with caution although evidence for little to no cardiotoxicity also exists. Sedative effects of all antihistamines should be considered and monitored during treatment particularly in instances where concurrent p450 metabolised medication is used. Systemic decongestants should be used with caution for their stimulatory and vasoconstrictive effects that may manifest as agitation and hypertension (with possible intracranial complications) respectively. Complicating and co-existent pathologies such as diabetes will induce nasal pathologies such as drug resistant *Hemophilus influenzae* (flu) infections which will complicate treatment of allergic rhinitis [24]. With non-allergic causes of rhinorrhea predominating the clinical landscape in this age group, new onset allergic rhinitis may be underreported and under diagnosed [25].

Overall performance status or mobility along with co-existent joint problems such as arthritis, particularly if affecting neck and small joints of the fingers, should be taken into account when prescribing medication that not only needs coordination but also accurate positioning for optimal delivery. As such, topical nasal drops which necessitate positioning of the head upside down, may be too difficult for some to accomplish thereby significantly

reducing compliance. Such difficulties were highlighted by the GINA [26••] recommendations in relationship to asthma medication and do very much exist in the context of intranasal treatment. Appropriate delivery will minimise ingestion of intranasal medication which may be as high as 70 % [27]. Choice of medication should be guided by knowledge of bioavailability of different medications with mometasone proprionate, fluticasone propionate and fluticasone furoate ideal candidates. Beclomethasone, the first choice in most primary care settings, has one of the highest systemic absorption. This, as well as the inevitable issues with delivery, makes beclomethasone a poor first line treatment in the elderly. Osteoblastic and osteoclastic activity may be altered with corticosteroid use but year-long use of fluticasone proprionate at 200 μg is not associated with osteoporosis [28].

Implication of polypharmacy in the elderly poses its particular concerns: possible aging related effects on memory will affect compliance with steroid use, which in turn may cross react with existing medication regiments [29]. Decreased renal function and drug clearance as well as diminuted hepatic metabolism alter pharmacokinetics and pharmacodynamics of medicines. Postural hypotensive and sedative effects of drugs should be particularly borne in mind when prescribing for the elderly. Poor receptor selectivity of first generation antihistamines endows this group of drugs with dopaminergic, serotoninergic, cholinergic and muscarinic properties that may exacerbate urinary retentions, tachycardia and postural drop. Co-existent conditions such as glaucoma, prostatic hypertrophy and hypertension (with anti-hypertensive medication) makes first generation antihistamines a poor choice in this age group. Second generation antihistamines are metabolised by the p450 hepatic system and excreted by the kidneys, which should be borne in mind in significant number of patients where hepatic disease or altered kidney function co-exist [30]. Cetirizine, ebastine, fexofenadine and levocetirizine may require dose alteration whilst desloratidine and rupatadine, in healthy subjects, should not [31].

Oral corticosteroid treatment for asthma is associated with side effects ranging from mild to severe including peptic ulceration [32], worsening of pre-existing diabetes mellitus (or development of diabetes itself) [33], memory impairment, osteoporosis. With good treatment outcomes achieved with topical corticosteroids with or without concomitant use of newer antihistamines, the side effects of oral steroid treatment outweigh any potential benefit and have no role in the treatment of allergic rhinitis in this age group.

There is increasing evidence in the literature for using immunotherapy in the elderly for allergic rhinitis. Sublingual immunotherapy (SLIT) does seem to be beneficial on those with perennial allergic rhinitis with bronchial asthma [34]. More recently, house dust mite SLIT was shown to be beneficial, as measured by reduction in medication use and improvement of symptoms scores, in the 60–75 age group in a double-blind randomised placebo controlled trial with no reported serious side effects [35].

Pregnancy and breast feeding

Rhinitis in pregnancy requires careful diagnosis. With all types of rhinitis capable of causing symptoms during pregnancy, it is important to differentiate between different pathologies, keeping in mind the unique rhinological entity

of pregnancy-induced rhinitis [36••]. Prevalence of rhinitis in pregnancy of variable aetiology may be as high 30 %. Rhinitis symptoms, like asthma, may worsen, improve or plateau during pregnancy. Establishing the allergic component of rhinitis in gravidas is crucial to direct therapeutic treatment. There is enhanced focus on conservative treatment options such as raising the head of the bed, saline douche and washes, as well mechanical enhancers of nasal airflow such as spring-loaded strips, mandibular advancement devices or even non-invasive ventilation such as CPAP. Comorbidity of worsening nasal airway may exacerbate pregnancy induced alterations in vascular turgor, aggravating the congestion. In severe cases, such clinical picture may tip over into obstructive sleep apnoea which is associated with hypertension (associated with development of pre-eclampsia) and low APGAR scores and birth weight.

Pharmacological treatment of allergic rhinitis in pregnancy is guided by balancing of risks to expectant mother and baby, with symptom severity and potential clinical benefit. Topical corticosteroids have level B or C FDA drug risk rating, meaning no teratogenicity in animals without evidence in human studies (B) or that there are no adequate human or animal studies/demonstrate adverse effects in animal but not human studies (C). Budesonide having been studied in an inhaled preparation in asthma, and shown to be safe in pregnancy, has been granted B rating with all other topical corticosteroids possessing a C rating. Anecdotally, topical treatments are used widely when symptoms are persistent and severe and, in cases of pre-existing and possibly complicating chronic rhinosinusitis, may be beneficial in preventing exacerbations and complications from the later. The American College of Allergy, Asthma and Immunology expresses preference over budesonide in topical steroid treatment during gestation [37].

No studies on use of oral corticosteroids for allergic rhinitis in pregnancy exist but given their well now potential side effect, their use in pregnancy should be limited to cases where allergic rhinitis is complicated by asthma and episodes of lower airway exacerbations [38]. In the context of the asthmatic gravida, use of oral corticosteroids did not relate to foetal or maternal side effects, but these findings were negated when severity of asthma was stratified [39]. Moderate to severe asthmatics did experience significant side effects. These may include congenital malformations, such as cleft lip, adrenal suppression and low birth weight, mean oral corticosteroids do not play a role in the treatment of allergic rhinitis in pregnancy as a matter of course.

Use of topical and oral decongestants need careful consideration and advice as they can be accessed with ease and an over the counter (OTC) treatment. With over 90 % of pregnancy women using OTC medication and up to 25 % specifically using decongestant preparations, it is important for general physicians to be well versed in their potential side effects [40]. The main constituent of decongestants ephedrine and pseudoephedrine do not demonstrate teratogenic properties. Despite this some earlier studies have linked use of pseudoephedrine to congenital malformations such as gastroschesis, small intestinal atresia and ventricular septal defects and hemifacial microsomia (36). Phenylephrine has been linked to eye and minor limb malformation. The retrospective nature of these studies, with small sample sizes, and multiple confounding factors limit the strength of conclusions from these studies. Conversely, some studies are able to demonstrate no risk to the foetus, risk of preterm labour or low birth weight. A large case control surveillance study has shown increased

risk with decongestant use [40, 41]. Such an effect may be via α-adrenergic stimulation of placental vessels causing vasoconstriction and possible hypoxia induced oxidative stress [42]. The authors do highlight that even with increased odds ratio, the incidence is still very small and recommend further studies for accurate risk stratification [43]. Topical applications carry with them the real risk of rhinitis medicamentosa which may happen with as little as once daily use. Systemic treatments will avoid such tolerance but risk agitation, tachycardia and insomnia.

Sodium chromoglycate has a well established safety profile and is frequently used in the paediatric and elderly populations (36). It offers good symptomatic relief from itching and sneezing and to date, has not been shown to have teratogenic properties. The use of the anti-muscarinic, ipratropium bromide, should be restricted to cases where clear rhinorrhea is a particular issue as it will not deal with any other symptomatic aspect of rhinitis such as congestion, itching or sneezing. As such its sole use in allergic rhinitis is limited.

No studies to date have looked at the use of the above medications in lactating mothers [44]. The American Academy of Paediatrics has reported no side effects on babies of lactating mothers using fexofenadine, loratidine and prednisolone. These drugs do not result in signs or symptoms in the infant or have an effect on lactation itself. With pregnant and breast feeding women, therapy must focus on how essential treatment may be, and by implication, the impact of symptoms. The drug with most favourable and well-established safety profile should be used at the lowest dose necessary for effective symptom control.

Athletes

Allergic rhinitis and its treatment in athletes requires a multifaceted approach. Athletes are a self, and environment, selecting group of individuals competing at or near limits of their physiology often at times of marked increase in pollen counts [45]. Such intense stain will carry with it an immunological impact that will be superimposed on any pre-existing allergic conditions. The later is very often accompanied by alterations and possibly pathology of the lower airways and both are further compounded by variable insight on the part of the athlete into the nature, cause and possible impact of their symptoms on performance. Intense and protracted exercise alters both the adaptive and immune profile. T and B cells, NK cells are decreased post exercise, with increase in inflammatory cytokines and in augmented IgA and IgM secretion [46]. Activation of the hypothalamo-pituitary and the sympathetic autonomic adrenal systems which further communicate and enhance cytokine production may favour a Th2 cytokine profile [47]. The prevalence of allergic rhinitis in athletes varies between studies from 16.8 to 56 %, with at least half of those reporting symptoms, not seeking medical treatment [48].

Prevalence of rhinitis varies widely depending on the sport pursued. Equine sport participants have the least and swimmers the highest incidence with particular attention needed in runners, skiers and boxers. Each sport brings with it particular challenges and effects on the nasal mucosa. It is likely that the low atopy in equine sports is due to self-selection at an early stage of participation. Those allergic and reacting to animal dander, hay, stable dust, wood or highly sensitive to particulate matter

with a non-allergic rhinitis response, are unlikely to be able to pursue the sport to high competitive levels. Swimmers on the other hand are in constant contact with water and will participate in their chosen sport up to 30 h per week. It is estimated that swimmers may inhale as much as a litre a week [49]. Pool water is disinfected with combination of chlorine gas or hypochlorite liquid, which in combination with water pH, temperature and salt content, will cause an irritative rhinitis which includes allergic type symptoms of sneezing and water rhinorrhea. A study conducted before and after nasal clip usage in symptomatic competitive swimmers and comparing these with non-competitive, asymptomatic controls, shows 44 % of patients to be allergic rhinitics with the rest a combination of neutrophilic rhinitis, rhinosinusitis and non-allergic rhinitis, with eosinophils and with or without mast cells (NARES and NARESMA). The neutrophilia in all groups decreased after use of noseclips and nasal airway resistance improved, demonstrating the immunological and performance impact of nasal mucosal exposure to water [50]. Skiers performing in the cold and at high altitude will have increased parasympathetic-mediated hypersecretion and capacitance vessel-induced nasal congestion, leading to increased resistance and augmented mucociliary transport. Runners demonstrate excessive initial sympathetic activity with initial improvement of nasal airflow to be followed by rebound and excessive enhancement of nasal secretions. Detrimental symptoms of rhinitis are not due to infective pathology but are driven by underlying rhinitis [51]. The AQUA questionnaire has been shown to be a reliable screening tool in these individuals [52]. Boxers will suffer repeated nasal trauma, affecting the nasal architecture that is further compounded by use of caustic material to stem bleeding. The result is a scarred and deformed nose, particularly at the nasal valve level with marked impact of nasal airflow and chronic alteration of nasal mucosal function. In each of the above instances, the already high prevalence of allergic rhinitis is further compounded by the particular effect of each sport.

The nasal response to exercise enhanced sympathetic α-adrenergic vasoconstriction and concomitant, near immediate, drop in nasal resistance by up to 50 %. In rhinitics, this effect is diminuted and may be further offset by the parasympathetic hypersecretions that accompany increased activity [53]. The collective effect of the above will imply early shift to mouth-breathing in rhinitic athletes, which will have its own impact on the lower airways due to inhaled particulate or noxious matter. Ozone, smoke, sulphur and nitrogen dioxide will induce sensitization and enhance Th2-mediated responses in atopic individuals. The co-existence of lower airways disease must also be considered and evaluated in each allergic rhinitic athlete as treatment must address both. With exercise-induced bronchoconstriction a feature in this group of patients, a comprehensive approach using both direct and indirect measures of ventilator function is required. Methacholine challenge and eucapnic voluntary hyperventilation, with and without β2 agonist to ascertain reversibility of obstruction, as well as spirometry and skin prick testing should all be included for a holistic assessment of these patients [54].

When considering treatment in athletes, physicians should acquaint themselves with the up-to-date list of banned substances in competition. Information can be found on websites of the World Anti-doping Agency (https://www.wada-ama.org/en) [55], the Global Drug Reference Online (http://www.globaldro.com/Home) and the official website of the Olympic movement (http://www.globaldro.com). In summary, antihistamines provide an excellent first line therapy and will combat immediate symptoms of allergic rhinoconjuctivitis. Second generation medication is preferable to minimise sedative effects, and those not reliant on p450 hepatic metabolism will further prevent drug interactions and the rare but serious complication of cardiotoxicity (fexofenadine, cetirizine, desloratidine). Antileukotrienes are helpful in cases of co-existent asthma but will do little on their own in the treatment of AR. Mast cell stabilisers are equally beneficial when a large conjuctival component to AR symptoms exists and no ban on their use is in effect. Topical decongestants may be a tempting short-term treatment but with rules now permitting only up to a maximum dose, athletes should be informed and advised to stop any phenylephrine-containing medication at least 24 h before competition [56]. Topical steroids form the mainstay of allergic rhinitis treatment and are permitted. Minimising systemic absorption whilst maximising symptom control should be the focus of therapy with mometasone and fluticasone furoate excellent first choice treatments [28]. Greater insight and better holistic approach on part of both the athlete and physician will improve symptoms and competitive performance [7, 57•] (Table 1).

Immunosuppression/HIV

The link between altered immunoglobulin, cytokine and T cell profile in acquired immunodeficiencies such as HIV has been long observed [58]. The precise nature and degree of such alteration has been less straightforward to establish. Skewing of cytokine profiles with decrease in Th1 associated milieu will bias expression towards a more Th2 favourable environment; a change that is accompanied by T cell anergy [59]. Similarly, primary immune deficiencies, which may be more common that traditionally thought, and present an allergic

Table 1. Summary of allergic rhinitis treatment options in athletes with World Anti-Doping Agency (WADA) rules on usage effective January 2015

Antihistamines	No ban	Pragmatic approach to balance sedative effects of first generation medication
Anticholinergics	No ban	Helpful when rhinorrhoea a particular feature
Antileukotrienes	No ban	Helpful adjunct
Mast cell stabilisers	No ban	Particularly beneficial with co-existent conjuctival symptoms
Topical steroids	Caution	Requires TUE: with nasal, buccal, skin usage permitted To combine with inhaled steroids if co-existent asthma (inhaled steroids require TUE)
Oral steroids	Ban	Banned in competition, requires TUE
Immunotherapy	No ban	Should not be administered immediately before or after competitive physical activity
Ephedrine/methylephedrine	Ban/caution	Banned in competition with pseudoephedrine permitted

phenotype [60]. Alteration in IgE, bronchial hyperreactivity and respiratory symptoms, suggest increased incidence in acquired immunodeficiency individuals [61]. ENT presentations in HIV seropositive individuals are overwhelmingly common with 80 % seeking medical help for an ENT-related complaint. Up to 70 % of individuals will suffer symptoms associated with rhinological disease. Incidence of allergic rhinitis in particular doubles in HIV-infected patients and may be as high as 87 % post infection. Antileukotrienes and antihistamines are excellent first line treatment with no contraindication in this patient group [62]. Indeed, there is limited evidence for immunological reconstitution following montelukast therapy in HIV [63] in a very small case series of three patients. Topical steroids with short half-lives and minimal systemic load should be used, bearing in mind possibility of iatrogenic Cushing's disease when administered with highly active anti-retroviral treatment (HAART) such as ritonavir.

General immunocompromise has been included as a contraindication to allergen immunotherapy (AIT) with recent evidence highlighting the need for specific examination of HIV and its relation to AIT [64••]. Sublingual immunotherapy (SLIT) has been recently shown to be safe and to improve clinical outcomes without altering CD4 subsets or viral load [65]. Individual assessment and treatment plan should therefore consider the use of AIT in acquired immune deficiencies with judicious evaluation of symptoms and their severity to maximise possibility of optimum treatment.

Comorbidities

Lower airway conditions as well as other rhinological pathologies, such as chronic rhinosinusitis/nasal polyposis, will have their individual impact on allergic rhinitis. Asthma is the commonest comorbidity with 80 % of asthmatics having AR and 30–50 % of allergic rhinitis patients having asthma. Moderate and/or persistent allergic rhinitis bestows an increased risk of poorly controlled asthma, a prediction that holds true regardless of age or geography [66]. Allergic rhinitic (versus non-rhinitic) asthmatic children experience increased frequency of wheezing episodes, which are more severe and in need of medical attention more often. Use of intranasal corticosteroids renders this difference nonsignificant [67]. The prevalence of allergic rhinitis in nasal polyp patients varies from 10 % [68] to as high as 64 % [69]. The prevalence of nasal polyps amongst allergic rhinitis patients is no different than the general population [70, 71]. Eosinophil counts in nasal polyps show correlation with specific and total IgE counts [72]. Evidence of atopy is present in 50 to 84 % of CRS patients [73, 74]. In contrast, atopic CRS patients do not have infective episodes when exposed to the sensitised allergen [75]. Allergic rhinitis driven nasal congestion results in microarousals and non-restorative sleep [76••]. Conversely, allergy is not a risk factor for development of CRSwNP [77].

Only 3.6 % of over 4000 allergic rhinitis sufferers surveyed as part of 'Burden of Allergic Rhinitis' report 100 % sleep quality with significant impact on quality of life, cognitive ability and school/workplace productivity [78] demonstrating the impact of AR on sleep [79]. This demonstrates the need for a holistic approach in treating allergic rhinitis, starting with sound

history to outline possible co-existent pathologies that may adversely affect treatment.

Conclusions

Treatment of allergic rhinitis relies on full appreciation of the multifaceted nature of presentation variable disease severity and co-existent pathologies which may also need addressing to achieve full symptom control. Physicians managing allergic rhinitis must acquaint themselves with the nuances of its treatment in specific conditions.

Compliance with Ethical Standards

Conflict of Interest
Dr. Nara T. Orban declares no conflicts of interest.

Human and Animal Rights and Informed Consent
This article does not contain any studies with human or animal subjects performed by any of the authors.

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