



REVIEW ARTICLE

Allergic rhinitis in children

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Allergic rhinitis affects up to 40% of children but is commonly undiagnosed. Careful assessment of nasal symptoms allows for the most appropriate therapeutic options to be chosen. Allergen avoidance is often difficult in practice. Antihistamines are of limited benefit in allergic rhinitis caused by house dust mite and other perennial allergens, where symptoms, predominantly nasal obstruction, are not histamine mediated. In contrast, symptoms triggered by pollen, such as nasal itch, rhinorrhoea and sneezing, are relieved by antihistamines. Intranasal steroids are the treatment of choice for persistent moderate–severe allergic rhinitis and are more effective than antihistamines for relief of nasal obstruction. Failure to respond to intranasal medications is often caused by poor compliance or inefficient use of nasal sprays. Immunotherapy may be a useful, if expensive, option, particularly where symptoms are because of a specific pollen. The benefits of immunotherapy in house dust mite-induced rhinitis and asthma remain controversial.

Key words: allergic rhinitis; child; rhinitis, diagnosis; rhinitis, therapy.

Allergic rhinitis (AR) affects 10–40% of children world-wide and is increasing in prevalence (Table 1).^{1,2} AR is commonly under-diagnosed, with symptoms frequently attributed to a recurrent cold. Parents may not seek medical attention or use medications available without prescription to treat symptoms suboptimally. AR frequently coexists with asthma (most studies report a rate of 50–60%).³ A Cochrane review has demonstrated that treatment of AR in adults and children with concomitant asthma can improve asthma control and reduce hospital visits.⁴ Untreated, AR can predispose to rhinosinusitis,⁵ otitis media and impaired hearing (secondary to Eustachian tube dysfunction),⁶ disordered sleep⁷ and adverse consequences for cognitive/school performance.⁸

Classification

Historically, AR has been classified as either perennial (PAR) or seasonal ('hay fever'), dependent on whether symptoms occur throughout the year or in relation to seasonal exposure to

allergen. Seasonal allergens include tree/grass pollens and fungal spores; allergens implicated in PAR include the house dust mite (HDM) (*Dermatophagoides pteronyssinus* and *Dermatophagoides farinae*) and animal dander; note, however, that in many countries including Australia, 'seasonal' allergens (e.g. plantain) may be present throughout the year. As a consequence, a World Health Organization workshop proposed a new classification in 2001, using frequency and severity of symptoms to guide therapy (Figure 1).

Pathophysiology and Its Relevance to Therapy

Allergen exposure in sensitised individuals induces the release of inflammatory mediators via an Immunoglobulin E (IgE)-dependent mechanism, resulting in the symptoms of AR as demonstrated in Figure 2. Ongoing allergen exposure induces nasal airway hyperresponsiveness (NAHR), a hallmark of AR. NAHR is a pathophysiological state whereby the response of the nasal airway to both allergen and mediators (such as histamine and bradykinin) is increased compared with normal.⁹ Thus, in experimental terms, the same level of exposure to antigen and other mediators results in increased nasal obstruction, tissue oedema and production of secretions. While a variety of pharmacological agents can be used to treat the initial inflammatory response, only steroids are effective in preventing NAHR. In patients with AR to seasonal allergens, maximum effect is achieved if steroids are commenced prior to the pollen season, before NAHR is established.¹⁰

Traditionally, AR has been considered to be a single disease entity, but different allergens may induce symptoms via distinct mechanisms.¹¹ Patients sensitive to pollen and other 'seasonal' allergens typically present with marked rhinorrhoea and sneeze. Antihistamines reduce these symptoms but have little effect on nasal blockage. Furthermore, antihistamines are of limited

Key Points

- 1 Antihistamines are of limited benefit in rhinitis caused by house dust mite and other perennial allergens, because symptoms, predominantly nasal obstruction, are not histamine mediated.
- 2 Intranasal steroids are the treatment of choice for persistent moderate–severe allergic rhinitis.
- 3 Treatment failure is frequently due to poor technique or compliance.

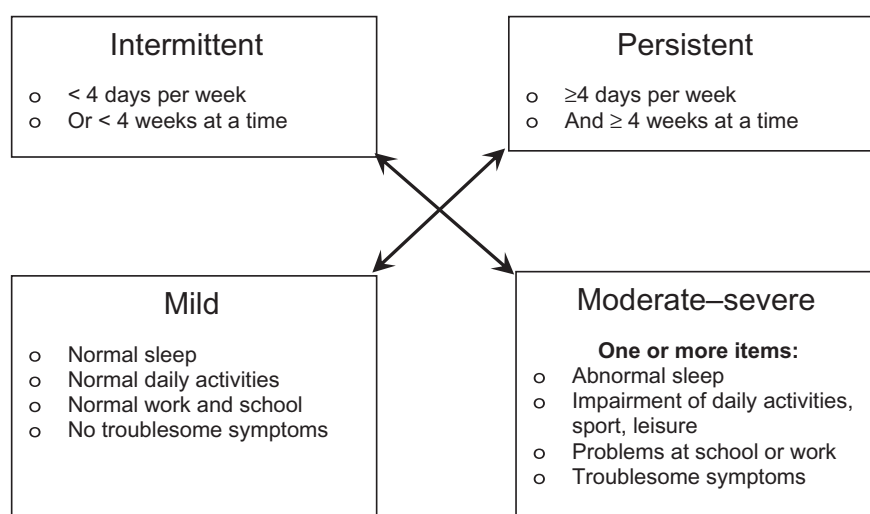
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Table 1 Prevalence of parent- or self-reported symptoms of rhinoconjunctivitis (defined as a problem with sneezing, or a runny or blocked nose in the absence of intercurrent infection together with eye symptoms) and hay fever

Symptoms of . . .	Age (years)	Worldwide (%)		Australia (%)	
		1997	2001/2002	1997	2001/2002*
Rhinoconjunctivitis within last year	6–7	5.9	14.9	12.0	12.9
	13–14	13.6	9.1	19.6	19.3
Hay fever (ever)	6–7	7.2	20.2	17.9	19.8
	13–14	16.4	12.9	26.7	47.7

Data extracted from the International Study of Asthma and Allergies in Childhood.^{1,2} *Data from Melbourne only.

**Fig. 1** Functional classification of allergic rhinitis proposed by the World Health Organization Allergic Rhinitis and Its Impact on Asthma workshop (2001).

benefit in HDM-induced AR, where the predominant symptom is nasal blockage (Table 2). This suggests that mediators other than histamine may be responsible for nasal obstruction. Indeed, HDM protein is able to directly stimulate the production of bradykinin and similar peptides.¹² Thus, a clinician might expect a poor response to antihistamines in patients where HDM is thought to be the primary allergen.

Diagnosis

Assessing the nature of nasal symptoms and whether these are persistent or linked temporally with exposure to a known allergic trigger will provide initial clues as to an allergic cause. Other relevant symptoms include mouth breathing, snoring, learning/attention problems and disturbed sleep; the impact of these on the child's quality of life should be determined.¹³ Unilateral or unusual nasal symptoms suggest a non-allergic aetiology (Table 3). It is common for there to be a discrepancy in nasal airflow between the nostrils because of the phenomenon of nasal cycling, where symptoms, predominantly nasal obstruction, alternate between nostrils every 3–4 h because of homeostatic variations in mucosal blood flow. However, persisting unilateral obstruction in a child should alert the physician to the

possibility of septal deviation or a foreign body. Failure to respond to previous therapy is helpful in guiding further treatment and may provide some diagnostic clues (e.g. lack of response to antihistamines in HDM-induced AR).

Observation of the child during the consultation may provide further clues: Mouth breathing can imply nasal obstruction, while a horizontal crease across the dorsum of the nose (caused by the 'allergic salute', an upward rubbing of the nose with the palm of the hand in response to nasal symptoms) supports a diagnosis of AR. Assessing nasal airflow by viewing condensation forming on a metal spatula or the handle of an otoscope placed under the nose can demonstrate unilateral obstruction or differential airflow. Direct visualisation of the nasal cavity using an otoscope will aid detection of purulent secretions, some anatomical abnormalities and nasal mucosal inflammation.

Investigations

Skin prick testing (SPT) can be helpful in determining an allergic cause and is now accessible to many paediatricians. SPTs have a high negative predictive value, so can be used to exclude an allergen as a possible cause. However, skin sensitisation only indicates the presence of specific IgE, and this may not translate

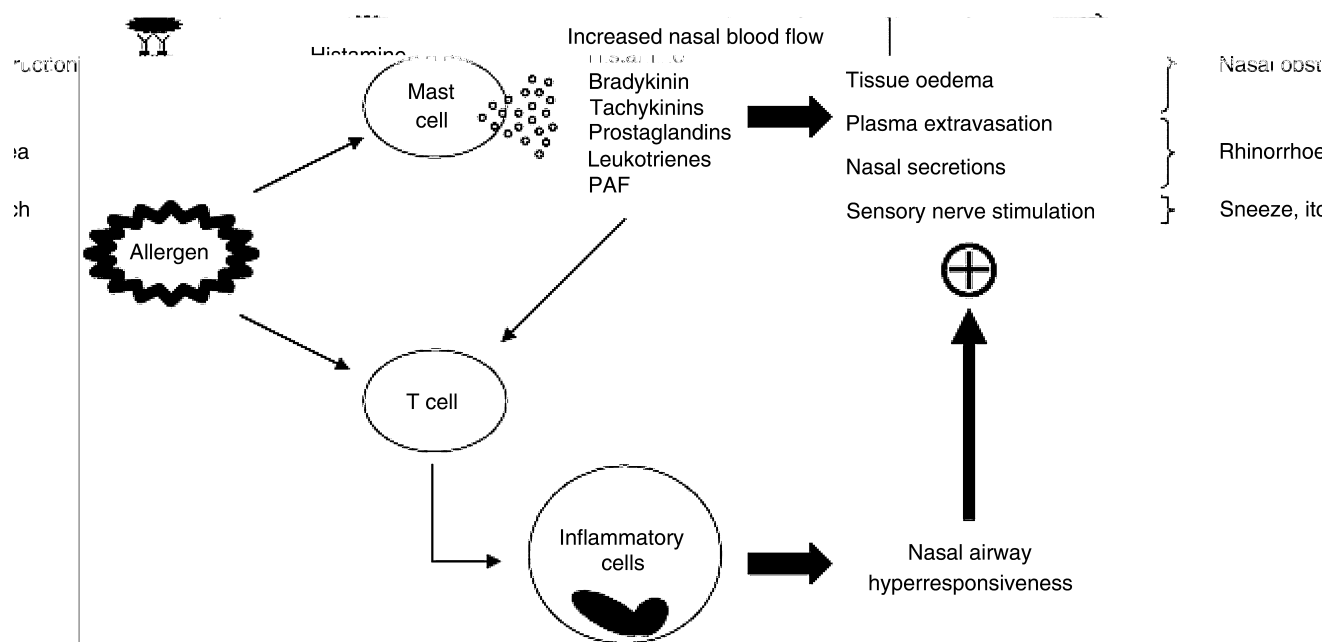


Fig. 2 Pathophysiology of allergic rhinitis. Initial allergen exposure in susceptible individuals results in the production of IgE antibodies, which become fixed to cells such as mast cells, a process known as sensitisation. Subsequent exposure causes the release of inflammatory mediators, generating an immediate, IgE-dependent allergic response. About one third of patients with allergic rhinitis experience a late phase reaction 6–12 h after exposure, associated histologically with an influx of inflammatory cells (including eosinophils, neutrophils and T-lymphocytes) to the nasal airways, where they release a variety of mediators that further exacerbate symptoms.⁹ IgE, Immunoglobulin E; PAF, Platelet activating factor.

Table 2 Features of HDM-induced AR compared with AR induced by pollen¹¹

Feature	HDM-induced AR	Pollen-induced AR
Symptoms	Nasal obstruction prominent	Nasal pruritus and sneezing, with relatively low nasal obstruction
Temporality	Perennial	Typically seasonal, though symptoms can persist throughout the year
Eye symptoms	Infrequent	Common
Mediators involved	Kinins > histamine	Predominantly histamine
Response to antihistamine	Poor	Good
Response to steroid	Good	Good

AR, allergic rhinitis; HDM, house dust mite.

into clinical symptoms. Up to 30% of children have a positive SPT to HDM, but only a proportion have associated symptoms.¹⁴ At least 15% of subjects fail to develop symptoms to an intranasal challenge with an allergen to which they are SPT positive.¹⁵ Therefore, it is essential to test only for those allergens that, in light of the clinical history, might be implicated. The indiscriminate ordering of ‘panels’ of large numbers of allergens is at best unhelpful, generating confusing results and adversely affecting families through inappropriate attempts at allergen avoidance. Measurement of specific IgE can be performed where SPT is not available, but is less sensitive than SPT for most inhaled allergens.¹⁶ Results must be interpreted together with the clinical history. A mismatch between symptoms and allergy

tests – for example, a child with symptoms in springtime but SPT positive only to HDM, an allergen which causes perennial symptoms – should prompt consideration of other non-allergic causes.

Specialist laboratories offer a number of objective methods to assess the nasal airway, including nasal endoscopy, rhinomanometry and rhinometry (which assess nasal airflow), exhaled nitric oxide (a marker of inflammation and/or nasal polyposis) and cytological evaluation (nasal smear, lavage and biopsy). However, their use in children can be challenging. Lung function may be considered in children with persistent rhinitis and able to comply with testing, in order to investigate for concurrent asthma.

Table 3 Non-allergic causes of symptoms of rhinitis²⁶

Disease entity	Symptoms/signs	Potential trigger
Infective	Episodes of rhinitis every 4–8 weeks of mucousy nasal discharge lasting 5–7 days.	Typically, viral infections. Also bacterial (rarely, fungal in immunosuppressed children).
Autonomic or vasomotor rhinitis	Sudden onset and offset of watery discharge.	Strong smells or changes in ambient temperature.
Eosinophilic or non-allergic with eosinophilia (NARES)	Perennial symptoms with paroxysmal episodes. Skin prick tests are negative.	50% develop aspirin-sensitive disease with asthma and nasal polyposis in later life.
Drug induced	Nasal blockage.	blockers, chlorpromazine, aspirin, topical decongestants.
Hormonal	Nasal blockage and/or rhinorrhoea.	Puberty, oral contraceptives
Primary mucous defect	Obstruction consistent with nasal polyps.	Cystic fibrosis
Primary ciliary dyskinesias	Rhinosinusitis, bronchiectasis (and reduced fertility in adulthood).	Kartagener, Young syndromes
Systemic/inflammatory	Nasal blockage, nasal crusting. Polyps, sinusitis, asthma, peripheral eosinophilia.	Autoimmune disease Churg–Strauss
Structural defects	Unilateral nasal obstruction.	Nasal septal deviation, adenoidal hypertrophy. CSF leak is a very uncommon cause of unilateral rhinorrhoea.

CSF, cerebrospinal fluid; NARES, Non-allergic rhinitis with eosinophils syndrome.

Management

Numerous options exist for the management of AR, most of which are available outside a specialist allergy service. Families require education as to the nature of the disease, benefits of treatment (including improved school performance and possibly asthma control) and the need for compliance with recommended treatments and their method of administration.

Allergen avoidance

Logic dictates that allergen avoidance should result in reduced symptoms; unfortunately, the degree of avoidance required may prove difficult or impossible to attain. Trials assessing HDM avoidance measures have proved disappointing, with a Cochrane review concluding that while some methods (such as the use of acaricides in combination with environmental control measures) may be helpful, the evidence for benefit is poor.¹⁷ Isolated use of bedding impermeable to HDM can reduce antigen levels by up to 70%, but this is probably insufficient to ameliorate symptoms in many patients. Combined measures using impermeable bedding, air filtration, replacement of soft furnishings and carpet may be more successful, but their use depends on the motivation of the family and their ability to afford the measures recommended. Some guidelines continue to recommend pollen avoidance strategies, including avoiding activities outdoors, indoor air conditioning and use of nasal filters. In fact, reasonable evidence exists only for the use of nasal filters,¹⁸ other interventions being based on expert consensus.¹³ In any event, their implementation in children can be very difficult.

Families will often enquire as to whether short-term removal of pets will improve symptoms sensitive to the offending dander. Unfortunately, it can take several months for allergen levels to

fall to levels which no longer provoke symptoms; thus, short-term measures are typically ineffective.¹⁹

Drug therapy

The pharmacotherapy of AR in children is similar in many aspects to that in adults but requires attention to dosing and the avoidance of adverse effects. A range of pharmacological options exist, as shown in Table 4. Topical application minimises the possibility of systemic effects, though tuition in the use of intra-nasal administration is essential, as therapy failure is often because of poor compliance and/or technique (see Fig. 3).²⁰

Intranasal corticosteroids

Intranasal steroids are the most effective treatment available for AR, and most guidelines recommend their use as first-line agents in children with moderate to severe symptoms.^{13,21} Two formulations are licensed in Australia for use in younger children: Avamys, GSK Ltd, Victoria (micronised fluticasone furoate) from 2 years, and Nasonex, MSD Ltd, New South Wales (mometasone) in children above 3 (though Nasonex is licensed in the USA for use in children above 2 years). Other preparations licensed for use in children over 6 years include Rhinocort, AstraZeneca Ltd, New South Wales (budesonide). Beclomethasone and fluticasone propionate are only recommended in children over 12 years, though both formulations are licensed in children over 6 years in USA and UK. Data from adult studies, many of which have included children, have clearly demonstrated both subjective and objective improvements in all nasal (particularly nasal obstruction) and ocular symptoms, with evidence to imply their superiority over oral and intranasal antihistamines.²² A Cochrane review published in 2007 specifically evaluating the use of intranasal steroids in children with AR was

Table 4 Effect of medications on symptoms of rhinitis in children

	Nasal pruritus	Rhinorrhoea	Nasal obstruction	Eye symptoms
H1-antihistamines				
Oral	+++	++	±	++
Intranasal	+++	++	+	–
Eye drops	–	–	–	++
Corticosteroids				
Intranasal	+++	+++	+++	++
Cromoglycate				
Intranasal	+	+	+	–
Eye drops	–	–	–	++
Decongestants				
Oral	–	–	+	–
Intranasal	–	–	+++	–
Leukotriene antagonists	–	+	++	++

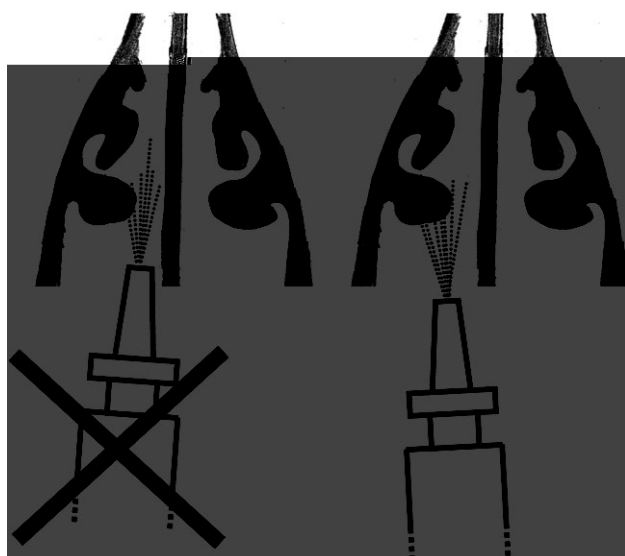


Fig. 3 Correct administration of a nasal spray, to optimise exposure of the mucosa overlying the inferior turbinate to the drug. Nasal sprays should be administered with the nozzle just inside the nose and directed laterally towards the outside wall, in order to achieve exposure of the nasal mucosa overlying the inferior turbinate to the drug. Children should be instructed *not* to sniff hard as this impedes the equal distribution of the solution and results in increased deposition in the nasopharynx.

severely limited by the exclusion of trials that included predominantly adults or permitted the use of rescue medication.²³ Nonetheless, a number of meta-analyses have provided robust evidence for their use in this age group and have demonstrated significant efficacy against nasal symptoms and, in particular, nasal blockage.^{24,25} Prophylactic use prior to the pollen season has also been shown to improve severity of symptoms,¹⁰ though intranasal steroids are not licensed for this use in children under 12 years.²⁶ While administration is recommended on a daily basis, there is also evidence to suggest that intermittent use of

beclomethasone or fluticasone on an 'as needed' basis over 4–6 weeks can also be effective.²⁷ Where nasal obstruction is significant, use of a saline nasal spray to clear secretions prior to steroid administration may improve drug delivery. Some clinicians recommend using a nasal vasoconstrictor spray prior to the administration of steroid to allow better entry of the steroid spray into the nasal air passages; however, their use can lead to rebound nasal congestion and, thus, should be used with caution (see below).

Parents and health professionals alike often raise concerns as to the consequences of steroid medication in children, with the potential for hypothalamic–pituitary–adrenal suppression, effects on bone mineralisation, growth retardation, and development of cataracts and glaucoma. The systemic bioavailability of fluticasone furoate (Avamys) and mometasone (Nasonex) are significantly lower than for other preparations (Fig. 4).²⁶ Although use of intranasal beclomethasone over a 1-year period had some effect on growth in children in one study, this has not been demonstrated with fluticasone or mometasone.^{28,29} Indeed, Möller *et al.* found no effect on growth or HPA axis function with children (aged 5–15 years) using daily budesonide for up to 2 years.³⁰ Current recommendations are to choose a steroid with low systemic bioavailability at the minimum dose required to achieve symptom control. Caution must be exercised in children receiving steroid medication for other indications, allergic or otherwise. Mild adverse effects (such as nasal dryness, blood-tinged secretions and headache) are common, though, interestingly, their incidence is not dissimilar to those reported for placebo. Nasal mucosal atrophy does not appear to be an issue, even with high-potency steroids.²⁶

Antihistamines

Antihistamines are effective for histamine-related symptoms such as itching, rhinorrhoea and sneezing, but not for nasal blockage, which is predominantly caused by other mediators.^{11,26} They can be used as first-line treatment for mild symptoms or in pollen-induced AR where nasal obstruction tends to be less of an issue. First-generation antihistamines (e.g.

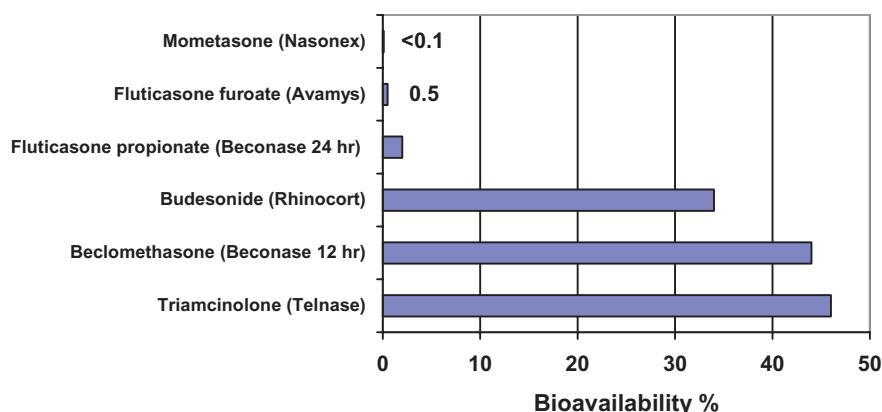


Fig. 4 Systemic bioavailability of intranasal corticosteroids available in Australia.

chlorphenamine and promethazine) are readily available without prescription. Unfortunately, they frequently causes adverse effects because of their ability to cross the blood–brain barrier. These include somnolence, impaired cognitive functioning and paradoxical reactions (restlessness, insomnia and, rarely, seizures), in addition to anticholinergic effects (dry mouth, blurred vision), which can be unpleasant.³¹ The use of newer ('second-generation') antihistamines (e.g. cetirizine, loratidine and fexofenadine), which have greater selectivity for peripheral H₁-receptors and do not penetrate the blood–brain barrier, should be encouraged. Furthermore, some of these newer agents appear to have anti-allergic effects independent of their action at histamine receptors. Many are now available as liquid formulations without prescription. Intranasal antihistamines are as effective as oral antihistamines and have a rapid onset of action; azelastine and levocabastine are licensed for use in children above 5 and 6 years, respectively.

Second-generation antihistamines are safe and well tolerated in children. Daily administration of cetirizine to children aged 12–24 months over an 18+-month period in a multi-centre study trial for eczema prevention revealed no significant adverse effects (including sedation) compared with placebo.³² Their main limitation tends to their lack of efficacy against nasal obstruction, particularly in PAR. Combined use of oral antihistamine with intranasal steroid does not appear to confer additional benefit over intranasal steroid alone, though Ratner *et al.* found that topical antihistamine in the form of azelastine nasal spray when combined with intranasal fluticasone was superior to treatment with fluticasone alone in seasonal AR.³³

Leukotriene receptor antagonists

Cysteinyl leukotrienes have been implicated in allergy airway inflammation, but the published evidence for the use of leukotriene antagonists in treating children with AR has been disappointing. Montelukast (Singulair, MSD Ltd, New South Wales) is the only leukotriene receptor antagonist currently licensed for use in younger children with AR (above 2 years) and has a good safety profile. Antihistamines appear to be more effective than leukotriene receptor antagonists in reducing nasal symptoms,^{34,35} and their use in addition to intranasal steroids does not confer any additional benefit.³⁵ In children with concomitant asthma, montelukast may be effective in treating both upper

and lower airway symptoms, particularly where the asthma appears to have a seasonal trigger.³⁶ A 1-month trial is usually sufficient to determine any treatment effect.

Systemic and topical nasal decongestants

These are very effective in relieving nasal congestion but are generally not recommended because of safety concerns. Decongestants, particularly oral agents, are associated with insomnia, irritability and poor school performance, and may induce cardiac events in some children.²⁶ Oral decongestants are now under increasing scrutiny across the globe; Australia's Therapeutic Goods Administration recently announced plans to follow regulatory authorities in the USA and UK in restricting their use in children because of lack of evidence of efficacy and concern as to rare but significant side effects.³⁷ As a result, they are not recommended for use in children or adults with rhinitis.¹³ Topical decongestants are unlikely to cause systemic effects and may relieve symptoms in the short term. However, their use beyond 5–7 days may lead to rebound congestion (rhinitis medicamentosa) and drying of the nasal mucosa, impairing the innate immunity of the nasal airway. They are therefore of limited value in the treatment of nasal congestion. Furthermore, families tend to continue the decongestant rather than limit use to a short course, as the child/family become accustomed to the open nasal airway and use the decongestant preferentially over other medications (which generally have slower onset of action). Many clinicians therefore advocate for the avoidance of these preparations altogether.

Immunotherapy

Specific immunotherapy is the only therapeutic option that modifies the immunological response to an allergen, rather than treating symptoms. It involves the exposure of a patient to small amounts of allergen in order to induce tolerance, and a number of different routes of administration have been investigated. Subcutaneous immunotherapy is effective in the treatment of AR in adults and adolescents,³⁸ but evidence is lacking in younger children.³⁹ Furthermore, suitability is limited by the need for multiple injections and the risk of serious or life-threatening reactions if the child has significant asthma, though in lower risk patients, systemic reactions are rare. A typical

treatment course requires monthly injections over a period of 3–5 years. However, the benefits of treatment may continue for many years following cessation. It may be recommended where there is a definite specific allergen trigger, the symptoms are severe and medications have been ineffective, inappropriate or have intolerable side effects.

Sublingual immunotherapy (SLIT) offers an alternative route of administration, which is more acceptable to paediatric practice. Allergens are administered in tablet or liquid form, ideally on a daily basis over 2–3 years. Minor oral reactions following SLIT are very common (including oral irritation, oral swelling and mild gastrointestinal symptoms), tending to resolve with the first few weeks of treatment. However, systemic adverse reactions are reassuringly rare (one study reported seven systemic reactions involving wheeze or worsening of nasal symptoms following 23 154 doses).⁴⁰ While a number of randomised controlled trials have shown SLIT to be effective in improving symptoms and requirement for medication in AR, others have not. Matters have not been clarified by published reviews and meta-analyses,⁴¹ which offer differing conclusions despite using similar data. Roder *et al.* performed a systematic review of published trials to mid-2006 and found 11 trials of SLIT in children deemed to be of high quality using the Delphi criteria. They concluded that there was no evidence of a positive effect and called for more randomised trials to establish efficacy.³⁹ However, the situation is confounded by significant methodological variations between studies, including type and dose of SLIT, polysensitivity between patients (SLIT may be more effective in those with fewer sensitivities), dosing frequency and duration, lack of preseasonal dosing (which increases efficacy) and wide variations in patient compliance (with some studies reporting a non-compliance rate of 20%).⁴¹ Nonetheless, almost all agree that SLIT is a safe and acceptable technique in children, with more evidence needed to determine efficacy and best practice. Two trials of SLIT in children with seasonal AR (with a total of over 500 patients) were published in early 2009, finding a significant improvement (median reduction of *c.* 30%) in both symptom scores and use of rescue medication.^{42,43} A recent systematic review (including these new studies) concluded that there is now reasonable evidence for the use of high-dose daily SLIT in children with AR to pollen, so long as treatment is commenced at least 4 months prior to the start of the pollen season.⁴⁴ However, evidence for the use of SLIT in patients with symptoms because of HDM remains equivocal. A significant limitation is the cost involved, both for the extract used as well as costs of administration in the case of subcutaneous immunotherapy.

An intriguing possibility is that immunotherapy might prevent the development of new sensitisation and progression to asthma. An open randomised study found that children who received SLIT reduced the onset of new sensitisations (on SPT) from 34% to 3% of subjects.⁴⁵ A second study followed up children 7 years after completing 3 years of subcutaneous immunotherapy to grass and/or birch pollen. The improvement in rhinoconjunctivitis and conjunctival sensitivity after immunotherapy persisted at follow-up 7 years later. In addition, fewer patients developed asthma (defined clinically as a recurrence of at least two of cough, wheeze or shortness of breath within a 12-month period) compared with controls (25% vs. 45%,

respectively), though the odds ratio of 2.5 (1.1–5.9) only just reached statistical significance.⁴⁶

Conclusion

Childhood AR is a common condition with significant morbidity. Careful assessment of the child with nasal symptoms allows for the most appropriate therapeutic options to be chosen, even in the absence of allergy testing. Education of the child and parent in the administration of the therapy is of critical importance. Immunotherapy, used appropriately, may offer significant advantages over traditional pharmacological therapies, but in most children, the latter can be used to successfully control symptoms until immunotherapy becomes more established in mainstream practice.

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Quiz

- 1 Which of the following is considered to best characterise the entity of persistent rhinitis as defined by the WHO?
 - A Rhinitis present at least 4 days per week
 - B Rhinitis present at least 6 days per week
 - C Rhinitis present outside the grass pollen seasons (spring and summer)
 - D Rhinitis present at least 6 days a week and for at least 6 weeks at a time

- E Rhinitis present at least 4 days a week and for at least 4 weeks at a time
- 2 With regard to allergen-induced rhinitis, which of the following is correct?
- A Grass pollens cannot cause perennial symptoms
 - B House dust mite-induced symptoms are predominantly nasal pruritus
 - C Antihistamines are very effective against house dust mite-induced symptoms
 - D Intranasal steroids are the treatment of choice for house dust mite-induced rhinitis
 - E Sublingual immunotherapy has been demonstrated to be more effective against rhinitis induced by house dust mite than that triggered by grass pollen
- 3 Concerning the use of intranasal steroids for che