

Seasonal allergic rhinitis – a review of current therapy

Lund VJ. Seasonal allergic rhinitis – a review of current therapy.
Allergy 1996; 51 (suppl 28): 5–7. © Munksgaard 1996.

Epidemiological studies in many countries strongly suggest an increase in the prevalence of seasonal allergic rhinitis, particularly in urban communities. The mechanisms of allergic rhinitis have been the subject of much research, elucidating symptomatology and improving the focus of therapeutic strategies. Ideally, avoidance of the offending allergen may be undertaken but is often incompatible with normal activity. Thus, active therapy is usually required, selected from a range of pharmacologic agents. Choice is determined by the severity of the symptoms, the age and individual circumstances of the patients, and may vary considerably from country to country. The menu will primarily include rapid onset, oral non-sedating H₁ antihistamines, topical nasal steroids and topical sodium cromoglycate to the nose, eyes or both. These medications may be given alone or in combination, usually under the direction of the primary care physician, and should be commenced in advance of exposure to the seasonal allergen. If this proves ineffective, specialist referral should be made for further management.

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Introduction

In the last one hundred years, we have learned a lot about seasonal allergic rhinitis and how it might be managed, but the treatment of 'hay-fever' remains open to improvement. The incidence of all forms of allergic rhinitis continues to increase, particularly in urban communities, implicating the environmental effects of our modern lifestyle (1). We would do well to reflect on the interesting observation made in 1873 by Charles Blackley, himself a hayfever sufferer, in his monograph (2) on the 'causes and nature of *Catarrhus Aestivus*': – that hayfever very rarely occurred in the working class but was a disease of the upper middle class.

The nose is the first line of defence against the assault of changing environmental factors on the respiratory tract, and the mechanisms of rhinitis have been the focus of much research, elucidating symptomatology and improving the basis of therapeutic strategies. Only by combining our knowledge of epidemiology with an accurate diagnosis of rhinitis and an understanding of the causative mechanisms can we hope to devise the most appropriate form of management for the individual patient.

Epidemiology

Traditionally, we divide our rhinitic patients into those who are affected during particular seasons and those who are affected all year round, but if patients have multiple allergy to seasonal allergens, they can readily end up with symptoms that can extend throughout the year. We are very well aware of the geographical variations that can exist, although our ability to explain them is less good. In general practice studies, differences have been shown between Denmark, where 11 per 1,000 GP consultations were related to seasonal allergic rhinitis, the UK with 20 per 1,000, and Australia, with the very high prevalence of 86 per 1,000. These figures, of course, will underestimate the genuine incidence and prevalence of the condition, because many patients will self-medicate and never actually go to their GP. In addition, the general practitioner may not always recognise the condition for what it is, whilst other factors such as cross reactivity between seasonal allergens will distort and exaggerate the problem. Again, the phenomenon of nasal priming (3) may make symptoms worse as the season progresses.

There is evidence from a number of sources that both seasonal and perennial allergic rhinitis are be-

coming more common in the general population, and especially in urban areas. A general practice study by Pedersen and Weeke (4) in the early 1980s showed a difference between the urban population of Copenhagen compared to the population in adjacent rural areas. A community-based study carried out in the USA by Broder (5) showed that in this particular group 75% of their patients with allergic rhinitis lived within the city as compared with those who lived in the surrounding countryside. There have been lots of suggestions as to why that increased prevalence may be occurring (1) and one that has attracted a great deal of interest, of course, is the issue of air pollution. In London, certainly, we are only too familiar with the problems that exist with increasing road traffic. How could that possibly be having an effect? It may be that the exposure of epithelial cells to substances such as ozone and nitrous oxide has a direct effect by increasing levels of cytokines such as GM-CSF (granulocyte/macrophage-colony-stimulating factor), interleukins and TNF (tumour necrosis factor). However, there are other, conflicting studies, such as the work of von Mutius comparing the incidence of childhood allergy in Leipzig and Munich (6). Despite the far higher levels of sulphur dioxide and particulate matter present in the air of eastern Germany, the prevalence of allergic rhinitis and asthma in Leipzig was found to be less than in the western city of Munich, suggesting that pollution may not be as relevant as we first thought.

Mechanisms

The familiar symptoms of seasonal allergic rhinitis – sneezing, watery rhinorrhoea and nasal itching – have allowed us traditionally to divide the patients with nasal allergy into ‘sneezers’ and ‘runners’, with nasal blockage characterising the more non-allergic conditions. How, then, are we going to manage this condition? Fig. 1 gives a diagram-

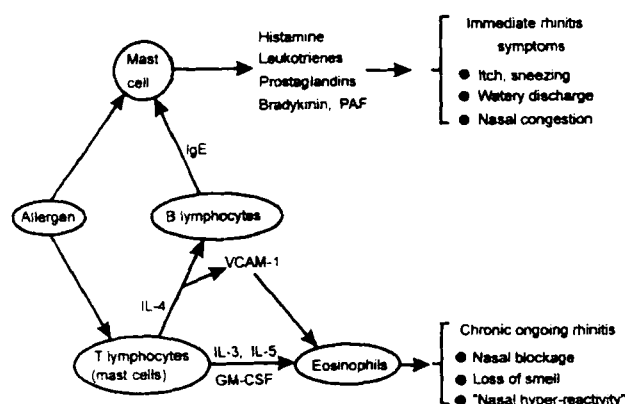


Fig. 1. Basic mechanisms involved in allergic rhinitis (from the 1994 International Consensus Report (7), with permission).

matic representation of some of the mechanisms responsible for allergic rhinitis in general, and clearly there are a number of points at which we can block this pathophysiological chain of events in order to abort the symptoms of rhinitis. In the first place, we can simply avoid the allergen. Secondly, we can affect the cells which are responsible for the release of the mediators – involving lymphocytes, mast cells and eosinophils – or, thirdly, having already got the mediators into the system, we can try and block them with medication. Immediate symptoms of rhinitis are caused by substances which are either released from granules (histamine; tryptase) or are newly formed (leukotrienes LTB₄, LTC₄; prostaglandin PGD₂) by the IgE-activated mast cells in the nasal mucosa. These mediators produce vasodilatation and increased vascular permeability, they increase glandular secretion, and they stimulate afferent nerve fibres, provoking sneezing and itching as well as the local release of pharmacologically active neuropeptides (tachykinins; substance-P). The ongoing process of allergic inflammation in the nasal mucosa leads to the accumulation of secondary inflammatory cells, especially eosinophils, which themselves release LTC₄ and toxic cationic proteins, damaging the mucosal epithelium and causing a chronic condition of nasal hyperreactivity associated with blockage and mucous rhinorrhoea.

– and management

There is quite a menu of drugs available for use in this area, but if we look at the therapeutic options and compare their ability to deal with the major symptoms of seasonal allergic rhinitis, there are obvious differences between the various drugs and their efficacy in this respect.

Mild disease/occasional symptoms

First, looking at the treatment of a mild condition and the patient who has intermittent symptoms, be they an adult or a child, we have various options to choose from: – allergen avoidance, the use of a second-generation non-sedating oral antihistamine, or the application of topical agents such as antihistamines or mast cell stabilisers to the eyes and the nose. Allergen avoidance is rather a counsel of perfection: it may be useful to know that the pollen count is rising or is likely to rise, so that we can institute therapy at an earlier stage, but clearly not every patient can avoid the allergens to which they know they react. Regarding antihistamine treatment, the systemic antihistamines are available in a considerable range, both in terms of their speed of onset of action and in terms of their dur-

ation of action, so we have quite a choice when it comes to dealing with the fairly mild seasonal condition, including cetirizine, terfenadine, astemizole and acrivastine. However, there seems to be a change in philosophy towards more topical medication as opposed to systemic medication. Obviously, a systemic antihistamine will have the advantage of dealing with both the nasal and the conjunctival symptoms, but we may be able to focus on the end organ with topical histamine antagonists such as levocabastine and azelastine. Sodium cromoglycate, a 'mast cell stabiliser' which prevents the release of histamine and other mediators from inflammatory cells, has been with us for a long time and it certainly has its place as a topical therapy. It has proved particularly useful in the special groups – children, pregnant women, and the elderly – but also for patients that are frequently affected, because its negligible side effects allow us to use it with considerable frequency and with impunity.

Moderate to severe disease

This should first be characterised as to whether nasal symptoms or eye symptoms predominate. If nasal symptoms predominate, then a topical nasal steroid would probably be our treatment of choice, and it is important that we start this medication 'ahead of the game' during the early part of the season, so that we have a possibility of pre-empting the accumulation of inflammatory cells and the release of their mediators. We can also add in the topical agents to control eye symptoms.

Decongestants and anticholinergics have limited activity and add little to the treatment of allergic

rhinitis. If eye symptoms predominate, our current options are the regular use of oral non-sedating antihistamines, or of sodium cromoglycate topically to the eyes, backed up by a topical nasal steroid. Finally, if these therapeutic strategies fail, specialist referral should be made for further management. Assuming there are no contraindications, we may then have to consider the use of short-term systemic corticosteroids, particularly when faced with a crisis situation. If the patient has only one or two specific allergens, we might also consider the use of immunotherapy. This recommended, stepwise approach to treatment (7) is undoubtedly our best bet for the management of allergic rhinitis, but as this short review of the current therapy demonstrates, the book is far from being closed.

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