

Diagnosing allergic rhinitis – is there a need?

A definitive diagnosis of allergic rhinitis makes a difference to management.

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Allergy is a hypersensitivity reaction initiated by immunological mechanisms. It can be IgE-mediated or non-IgE-mediated. Atopy is the inherited tendency to produce increased amounts of IgE in response to small quantities of allergen, which can produce clinical syndromes.

These allergic diseases or syndromes are common and include eczema, asthma, rhinitis, conjunctivitis, food allergies and others.

Rhinitis is the commonest manifestation, with a prevalence rate of between 4.5% and 38%. In South Africa the prevalence rate in teenagers is 28%.¹

It is accepted that both asthma and rhinitis are under-diagnosed. This may be due to limited access to health care, but without due suspicion under-diagnosis will continue. This then leads to exacerbations and complications.

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There is a belief among many practitioners that it is not necessary to establish an accurate diagnosis and that it is not cost effective. The result is that not only is the patient treated in an indecisive manner, but no attempt is made to explain the underlying mechanisms of the chronic condition, while the patient is expected to comply with daily medication which may be expensive. It is not surprising that treatment is often used incorrectly and may be inappropriate. Furthermore, management may be incomplete as environmental factors cannot be taken into consideration nor, certainly, can desensitisation be contemplated. In many instances patients are told to get rid of beloved pets without proof of causation.

There should be a different attitude to treating atopic as opposed to non-atopic rhinitis. An accurate diagnosis justifies treatment or the inappropriateness thereof, in which case therapy can be stopped and further investigation undertaken. Often a small child is prescribed multiple anti-allergy medications when the diagnosis

is repeated upper respiratory tract infections or obstruction due to adenoids or tonsils. Other diagnoses need to be considered as well.³

The diagnosis of allergic rhinitis should be approached under three main headings: the history, the examination and specific testing.

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History

The history must be detailed. It may be helpful to make a list of symptoms and go through them methodically, as many clues can be established in this way.

Besides the symptoms of rhinorrhoea, sneezing, itch, nasal congestion, postnasal drip and decreased sense of smell, trigger factors and relieving factors should be established. The allergic march is well recognised and concomitant symptoms of itchy, tearing eyes or asthma and certainly eczema add considerable evidence of underlying atopy. Eczema is the earliest manifestation of allergy in infants. Questions on possible complications must be asked. These include otitis media, diagnosed sinusitis, increased severity of concomitant asthma and significantly impaired quality of life, including poor sleep quality. Often the patient presents only with the complaint of 'sinus'. This should alert one to possible rhinitis. The concept of the united airway is also being accepted and all patients with asthma should be investigated for rhinitis.

Family history is important. If parents or siblings have atopic disease the likelihood of symptoms being due to allergy becomes significantly higher. It is important to ask specific questions, as many parents have not had their allergic diseases diagnosed, particularly in black families, where repeated respiratory infections become the accepted diagnosis. Antibiotic abuse is common when patients are treated for infections and not for allergy.

Systematic history cannot be neglected. Drug history should be established.

Past history may confirm that complications have already occurred, e.g. otitis media in young children with insertion of grommets, or a definite diagnosis of sinusitis with surgery.

Allergic rhinitis

Habits and response to treatment must be queried.

Environmental history is essential. Pet exposure and mould in the home can often clarify the origin of specific allergies. It is common for patients to refuse to accept the relationship of their symptoms to pets and an accurate diagnosis may convince them of the need for some avoidance. The relationship of symptoms to the patient's environment may be helpful. A recent case was solved by determining that the allergy involved was mould. Investigation revealed significant mould infestation problem in the home. Inland or coastal aggravation of symptoms helps to define which allergens are more probable.

It must also be borne in mind that in South Africa, and particularly in the grasslands, symptoms may not be seasonal as the pollen season is so prolonged. House dust mite sensitivity is associated with persistent symptoms.

At the end of the history the diagnosis of allergic rhinitis can often be made with a meaningful degree of certainty.

Examination

Examination may be helpful with evidence of features of rhinitis such as the long pale face of the mouth-breathing child, Morgan Dennie's lines, nasal crease, shiners, the allergic salute or constant rubbing of the nose, Hertoghe's sign (hypodense lateral eyebrows, dry mouth and lips and mouth breathing) (Figs 1-3). Typical mannerisms should be noted. Nasal turbinates may be glistening and prominent.⁴



Fig. 1. Long pale atopic face. (Photograph reprinted with permission from Du Toit G. *Clinical allergy images: the atopic syndrome*. Current Allergy & Clinical Immunology 2005; 18: 22-23.)



Fig. 2. Allergic shiners and Morgan Dennie's lines. (Photograph reprinted with permission from Du Toit G. *Clinical allergy images: the atopic syndrome*. Current Allergy & Clinical Immunology 2005; 18: 22-23.)



Fig. 3. 'Allergic salute'. (Photograph reprinted with permission from Du Toit G. *Clinical allergy images: the atopic syndrome*. Current Allergy & Clinical Immunology 2005; 18: 22-23.)

Special investigations

The history and the examination may be sufficient to make the diagnosis. However, in some patients, and more so in the very young, the diagnosis may be elusive and tests become the deciding factor. These can confirm the diagnosis and justify advice on environmental changes and the use of chronic medication.

Although theoretically the relationship between allergen exposure and clinically relevant symptoms can be confirmed only by a controlled challenge, the presence of a positive test with the positive clinical history is accepted as being proof of allergic disease.⁵ Should the tests be negative there is merit in stopping treatment and re-appraising the patient and the diagnosis.

There are several screening tests for markers of inflammation such as Hansel's stain for nasal eosinophilia and measurement of eosinophilic cationic protein and others. These are not advocated in day-to-day

practice. The tests for detection of allergen-specific IgE antibodies are those that should be done.

Skin prick testing (SPT) is the gold standard.^{6,7} It is simple and quick and relatively inexpensive. Patients of all ages can be tested, even infants. Where allergic disease is suspected, there is a need for testing. It is extremely safe. Contraindications to carrying out this test are the recent use of antihistamines or extensive eczema. A panel of inhalant allergens can be used. They are commercially available and are strictly standardised.

The resultant wheal and flare are an excellent way of demonstrating to the patient what happens in the nose in an acute attack. Patients appreciate the time taken to discuss their allergies and possible ways of avoiding exposure. Compliance with treatment is always a challenge but when patients see the test result, they accept far more readily the need to use therapy. It is hard enough to convince a patient that their pets may be responsible for their own or their children's problem, but with a positive test this may be convincing.

Finally, if desensitisation is being considered an accurate diagnosis is vital. There may be reactions to both grass pollens and mites and the history and the test results can determine which vaccine to use.

In vitro testing includes measurement of total IgE, which has a poor predictive value. It need not be used for routine testing.⁵ However, if SPTs are negative and allergic disease seems likely, a raised IgE may indicate an atopic state in which the allergens are unidentifiable and a trial of treatment is justified. Total IgE is not often significantly raised in inhalant sensitivity, but if it is high it may suggest food allergies.

In vitro testing includes the Cap Radio-Allergosorbent (RAST) technique of testing and results correlate well with SPTs. The Pharmacia Differential Atopic Test (Phadiatop) includes a range of inhalant allergens, namely grass, weeds, trees, moulds, animal epithelia and house dust mites. It is an excellent screening test. If positive, the individual tests can be done. The values should be greater than 0.35 KU/l as clinical relevance falls at lower levels. The higher the levels are, the stronger the likelihood of clinical disease.⁵ It should be noted that levels do not necessarily equate with severity of symptoms. Tests required should be stipulated, not ordered indiscriminately. It is this lack of specificity that makes testing expensive.

The question of food testing in AR is debatable. The main value may be in excluding foods as being responsible for symptoms, rather than incriminating them.

Milk, for example, is often blamed for symptoms and yet it is the least common allergen when foods are tested.

The food mix fx5 Cap RAST is available. These test results are much more difficult to interpret than the inhalant tests. Many a patient has been put on a diet without justification because of reported positivity, with no interpretation in the clinical setting. The work of Hugh Sampson in establishing predictive values for clinical relevance is extremely useful. Thus it has been advocated that in infants over 2 years of age a value for milk allergy should be more than 15 KU/l, for peanuts 14 KU/l and for wheat 26 KU/l. This would give a positive predictive value of 95%.⁸

There may be exceptions with strong clinical evidence of food allergy when values are low. However, the majority of patients reveal no history of adverse reactions, yet based on positive tests with low values, diets are introduced. All results should be interpreted in a clinical setting.

There is significant cross-reactivity between pollens and certain fruits, grains and vegetables. Patients may complain of an itchy palate and throat on eating some foods. This is due to cross-reactivity and hence the positive RAST tests.

Skin prick testing (SPT) is the gold standard.

The message is that positive reactions in food RAST testing should be carefully considered before suggesting avoidance. The gold standard is still the food challenge test. Although food allergy can be important in some individuals, and particularly young children, the role in allergic rhinitis is usually exaggerated.

Other *in vitro* tests are mainly research-orientated but the CAST test is becoming useful in allergy that is non-IgE-mediated, such as may occur with preservatives and food additives. They are expensive and should not be ordered indiscriminately. Interpretation can be difficult and it is recommended that such tests should be discussed with allergists or the laboratory.⁹

Treatment

The establishment of an accurate diagnosis of AR has significant benefits. The patient can be given an explanation of the disease, and allergen avoidance becomes justified. Exacerbations can perhaps be avoided, especially in the workplace. Compliance

with treatment improves and costs can be contained in that appropriate medication can be prescribed rather than the hit-and-miss attitude that prevails. Desensitisation can be advocated with certainty.

The mainstay of treatment is antihistamines and topical corticosteroids. The long-acting antihistamines should be prescribed. There is no place for the older generation, sedating drugs and combination of these with corticosteroids is definitely unacceptable, even for exacerbations. Use of antihistamines in children under the age of 2 years is not registered, but should an accurate diagnosis be made, use becomes justified. When the long-acting medication is not available (public hospitals) the use of short-acting antihistamines is preferable to no treatment.

Steroid sprays are safe and effective. They need to be used correctly and in the prescribed daily dose. Patients need reassurance on safety. Should symptoms be severe, a short course of oral steroids is justified and often leads to improvement, which can then be maintained with the topical medication. Decongestants are not to be used for allergic rhinitis.

If there is associated asthma or poor response, the leucotriene receptor antagonists can be tried. A course should be prescribed and the patient should be asked about the relative benefits. Reflux is often considered to be an aggravating factor for rhinitis. Treatment should be prescribed only if the history warrants it.

Desensitisation is the only possible cure. It requires careful choice of vaccine and method of use, either subcutaneous or sublingual. Patients need to accept that it is at least a 3-year commitment, that it is relatively expensive and not covered by medical aid although recognition of this therapy is constantly being sought. It has to be ordered on a name basis through the Medicines Control Council. Repeats need new applications every 6 months. Patients need education in accepting that results may not be dramatic or immediate. However, results can be excellent.

The diagnosis and successful management of allergic rhinitis is attainable if practitioners apply the guidelines as outlined above.

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In a nutshell

- Allergy is a hypersensitivity reaction initiated by immunological mechanisms.
- It can be IgE-mediated or non-IgE-mediated.
- Atopy is the inherited tendency to produce increased amounts of IgE in response to small quantities of allergen, which can produce clinical syndromes.
- These allergic diseases or syndromes are common and include eczema, asthma, rhinitis, conjunctivitis, food allergies and others.
- Asthma and allergic rhinitis are under-diagnosed.
- Allergic (atopic) rhinitis is now defined as being either intermittent or seasonal (IAR or SAR) or persistent (PER or PAR).
- There should be a different attitude to treating atopic as opposed to non-atopic rhinitis. An accurate diagnosis justifies treatment or the inappropriateness thereof, in which case therapy can be stopped and further investigation undertaken.
- The history must be detailed. It may be helpful to make a list of symptoms and go through them methodically, as many clues can be established in this way.
- In some patients, and more so in the very young, the diagnosis may be elusive and tests become the deciding factor.
- Skin prick testing (SPT) is the gold standard.
- The mainstay of treatment is antihistamines and topical corticosteroids.