

Rhinitis due to food allergies: fact or fiction?

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Abstract

Food allergy is often thought to be a cause of rhinitis by patients, whose views are frequently influenced by information obtained from the Internet. The incidence of food allergy in children is 2–8 per cent, although a vast majority will grow out of it, and food allergy is actually very rare in adults. Food allergy usually presents with multi-system involvement, most commonly cutaneous and gastrointestinal symptoms. Food allergy induced rhinitis is less common, and isolated rhinitis due to food allergy is extremely rare. Treatment for rhinitis due to food allergy is therefore rarely indicated.

This review summarises the literature related to the incidence, diagnosis and management of food allergy and food allergy induced rhinitis.

Key words: Food Allergy; Rhinitis; Cow's Milk Allergy

Introduction

A myriad of websites^{1–4} advise patients about food allergy, particularly cow's milk allergy, as a possible cause of allergic rhinitis. Many people believe that their nasal symptoms are due to food allergy. However, on specific allergy testing, they do not actually demonstrate such an allergy.^{5–11}

This article reviews the available evidence on food allergy causing rhinitis, with regard to its prevalence, diagnosis and management. The rationale for food allergy tests in the management of allergic rhinitis is examined, as is the case for advising food item avoidance or restriction.

Pathophysiology of rhinitis in food allergies

Food allergy, hypersensitivity and food intolerance are terms often used loosely to denote the entire spectrum of immunologic and non-immunologic manifestations of food intake associated disorders. In clinical practice, the use of these various terminologies may cause confusion and is often unhelpful. In this article, the use of the term 'food allergy' implies one of the immune mechanisms outlined below.

Four types of immunologic mechanisms may underlie sensitisation to foods and result in the clinical manifestations of allergy.¹² Type one hypersensitivity is immunoglobulin E (IgE)-mediated hypersensitivity. Type two hypersensitivity is antibody-dependent, cell-mediated cytotoxicity. In type three hypersensitivity, antigen–antibody–complement immune complexes are deposited in tissue, causing tissue damage. Finally, type four

hypersensitivity is mediated by T-lymphocytes. Type one (or IgE-mediated) hypersensitivity has been estimated to constitute only 5 per cent of proven food allergies.¹³ However, delayed immune reactions¹² (types two, three and four) may occur in combination and are responsible for the vast majority (95 per cent) of food allergies.¹³

Non-immunologic reactions to food may involve different mechanisms and may mimic true allergy. Neurogenic phenomena are responsible for gustatory rhinitis, which involves watery rhinorrhoea after eating hot and spicy foods.^{14,15} Psychosomatic or emotional factors associated with food intake may also be responsible for rhinitis in some patients, probably due to autonomic stimulation.¹⁶ Table I summarises the various mechanisms responsible for rhinitis associated with food. Although oral ingestion is the primary route of exposure to food allergens, the symptoms may rarely occur from direct inhalation of aerosolised particles containing allergenic food.^{17–20} Enzyme intolerance abnormalities such as lactose intolerance and dietary disorders are distinct conditions and should not be confused with food allergy.

Food additives and rhinitis

The first case of perennial rhinitis induced by food additives was reported in 2001.²¹ Since then, two double-blinded, placebo-controlled case series, both published in 2002,^{22,23} have provided evidence that only a small proportion (estimated to be 5 per cent) of patients diagnosed with perennial non-allergic or vasomotor rhinitis actually have food additive

TABLE I
MECHANISMS OF FOOD RELATED RHINITIS

Mechanism	Reaction type	Clinical aspects
Immunologic	Immediate (IgE-mediated or type I hypersensitivity) (Anaphylactic) ~5%	Within minutes Occasionally fatal Associated with wheezing, angio-oedema, oral & throat symptoms Most commonly due to egg, milk, peanut, soya, fish & shellfish History of atopic dermatitis commonly present Mechanism thought to be fixation of circulatory IgE in nasal tissues Rarely, food allergens directly reach nasal lining May also present as late-phase allergic reactions after 4–8 h Responsible for vast majority of seasonal and perennial food-associated rhinitis Type II: cytotoxic; IgG/IgM-mediated Type III: IgG complex mediated Type IV: cell-mediated, due to T-lymphocytes Chronic symptoms Often associated with corn, wheat, cow's milk, soya, yeast & acidic fruit (apple, kiwi, pear)
	Delayed (Types II, III & IV hypersensitivities) ~95%	
Non-immunologic	Neurogenic Psychosomatic Additive-induced	Usually after hot & spicy foods – 'gustatory rhinitis' Often associated with autonomic stimulation Food colorants and preservatives thought to be responsible for non-immunologic rhinitis in $\leq 3\%$ children & $\leq 2\%$ adults

Ig = immunoglobulin

intolerance. The aetiology is unclear but thought to be non-immunogenic. However, antihistamine treatment is effective, implying that histamine is somehow linked to pathogenesis.

Food allergies and allergic rhinitis

Prevalence of food allergy. Food allergy is most prevalent in the first one to two years of life, following the child's first exposure to dietary proteins through a variety of items in the diet. The prevalence of food allergy in children in the first year of life is estimated to be 6–8 per cent.²⁴ This figure falls rapidly to about 1–2 per cent in late childhood, due to the development of tolerance, and remains stable thereafter through to adulthood.

Young and colleagues studied an English population by mailed questionnaire and subsequently performed food challenges to investigate those who complained of food-related symptoms. They found the prevalence of food allergy to be 1.4–1.8 per cent for the total population, and they estimated a 6–8 per cent prevalence in the paediatric population.⁶ A study in the USA found that 33 per cent of those questioned expressed complaints of food allergy¹⁰ that resulted in restricted diet. Such elimination diets are often introduced without medical consultation.^{25,26} This may result in an unbalanced diet, with the potential risk of malnourishment.^{26–30}

Prevalence of food allergy induced rhinitis. It is difficult to estimate the true prevalence of food-induced allergic rhinitis, for a number of reasons. Firstly, rhinitis is invariably part of a constellation of symptoms and signs associated with food allergies, including asthma, eczema, oral allergic manifestations, urticaria and gastrointestinal symptoms. Isolated rhinitis due to food allergy is very rarely

reported. Secondly, there is often a significant rate of cross-allergenicity, with respiratory allergens causing rhinitis. Prior sensitisation with inhaled pollen proteins may result in cross-reactions with homologous fruit and vegetable proteins.⁷

A recent study from the Isle of Wight assessed a birth cohort of 1218 children. It demonstrated that, in 2.9 per cent of the children, egg allergy had a statistically significant correlation with increased rhinitis and asthma at four years of age.³¹ In an earlier study, rhinitis accounted for about 70 per cent of respiratory symptoms observed in children who underwent double-blind, placebo-controlled food challenges.³² Typically, rhinitis occurs in association with other manifestations of food allergy, such as cutaneous and gastrointestinal symptoms. The same study also found that rhinitis induced by oral food challenges occurred more frequently in infancy and early childhood compared with adulthood. Separate studies have estimated the prevalence of rhinitis in children with known cow's milk allergy to be between 21³³ and 39 per cent.³⁴ The prevalence of rhinitis in older children allergic to nuts, milk, egg or soya bean was found to be between 31 and 39 per cent in a placebo-controlled study.³⁵

Several studies^{5,6,10} have shown an approximately ten fold mismatch between the number of patients (or parents) reporting food allergies and those diagnosed by physicians. Roehr *et al.*³⁶ studied randomly selected children and adolescents in Berlin; they found that, out of the 61.5 per cent of 739 responders who initially reported food allergies, only 4.2 per cent were confirmed by food challenges. All individuals with severe reactions had previous atopic disorders. The reason for such discrepancy may be twofold. Firstly, there is a common tendency for individuals to associate a number of adverse symptoms with food. Secondly, there may have been genuine

food allergies in early life which had been lost through tolerance in later life, but the patient and their family continued to associate them with unrelated symptoms.

Common food allergens

Population studies^{37,38} in the West investigating the pattern and distribution of food allergies in young children have confirmed that the four major allergens are cow's milk, hen's egg, wheat and soya protein. Studies in the USA³⁹ and France⁴⁰ have shown that peanut is emerging as a fifth major allergen.

Cow's milk allergy has been the most extensively studied allergy in the paediatric age group. The vast majority of children develop tolerance to milk allergy by the age of three years, and the practice of hydrolysing milk proteins in feeds further reduces the antigenicity of milk protein. Most studies^{41,42} have shown that children with non-IgE mediated milk allergies outgrow their allergy faster than children with the IgE-mediated variant. Multiple food allergies are common in children, and many children who demonstrate cow milk allergy will also be allergic to other common food allergens.⁴²

There are fewer studies in adults. A recent study⁴³ on 286 Finnish university students concluded that food allergies were most commonly reported in those with current (73.2 per cent) or past (66.3 per cent) atopic dermatitis and asthma (63.8 per cent). The authors found that 44.8 per cent of subjects reported food allergies not associated with other atopic history. The commonest allergic reaction was oral allergic syndrome (which affected 51.2 per cent of students and presented as the solitary symptom in 36.7 per cent), followed by gastrointestinal symptoms (23.5 per cent) and worsening of atopic dermatitis (11.4 per cent). Rhinitis was reported in 5.7 per cent (never in isolation) and asthma in 4 per cent. Kiwi fruit, shellfish, tree nut, peach, banana, avocado and celery nearly always caused oral allergic syndrome, whereas rhinitis and asthma were caused mostly by fish and shellfish. Chocolate, beer, paprika, egg, plum, tomato and carrot were other reported allergens. However, there was generally a poor correlation between reported food allergies and skin prick or RadioAllergoSorbentTest, except in cases of fish allergy. In patients with a negative history of food allergy, tests were nearly always negative. Milk allergies (after ruling out lactose intolerance) nearly always produced gastrointestinal symptoms.

Diagnosis and treatment of food-induced allergic rhinitis

The public perception of food allergies is likely to result in far more complaints of this condition than can be clinically confirmed. An accurate, food-focussed history is of paramount importance.

Rhinitis as a solitary symptom, without involvement of other systems (i.e. oral, gut, lungs and skin) is almost never due to food allergy and does not merit any further investigation. Most adults complaining of rhinitis without a history of specific food

allergies or other atopic symptoms will almost always prove negative for food allergies and also do not warrant further investigation.

A history of anaphylactic reactions is important, particularly if the patient reports that symptoms presented within minutes or hours of ingesting a particular food item. In such cases, it may be necessary to perform further diagnostic tests to isolate the allergen, and patients should be advised strict avoidance. The majority of adults with food allergies will have a lifelong history of allergy, and it is important to document the childhood history of allergies and atopies.

In young children presenting with rhinorrhoea, often with a history of other atopies, the severity of symptoms will guide the necessity for further investigation and treatment. With mild symptoms, it is reasonable to reassure the parents that the vast majority of children will outgrow their allergies within a few years, and observation is a sensible option. In the presence of more severe symptoms, further tests may be appropriate. Skin (intradermal) prick tests, IgE assay and RadioAllergoSorbentTest (despite the fact that only a minority of food allergy is IgE-mediated) are the most commonly available tests. Specialist allergy clinics may also perform food challenge tests, with facilities available for resuscitation. In children, the commonest five allergenic foods should always be tested for in the first instance: cow's milk, wheat, soya, hen's egg and peanut. In patients with long standing food allergy related histories, it is often quite difficult to make an accurate diagnosis.

Many patients carry out selective dietary elimination tests before seeking medical advice, which should be continued for at least two weeks before any positive or negative conclusions can be drawn.⁴⁴ There can, of course, be various other underlying causes for allergic rhinitis,⁴⁵ and treatment, in the form of dietary elimination, is frequently unnecessary.

In common with other forms of allergic rhinitis, the mainstay of treatment for food-induced allergic rhinitis is antihistamines and topical steroids. It is also important for the clinician to be aware of exclusion diets and substitutes (e.g. soya milk and peanut oil free preparations). In reality, specialist advice from allergy clinics may be required for the few patients whose food-induced rhinitis requires treatment.

Conclusion

Although food-induced allergic rhinitis is common in young children, it is invariably one of a number of symptoms. The vast majority of children will outgrow their food allergy in a few years, although in a minority the symptoms will persist until adulthood. However, it is important for the clinician to appreciate that, in adult patients, the great majority of complaints of food allergy cannot be clinically verified.

Very rarely, cases of IgE-mediated food allergy, with a risk of anaphylaxis, will be encountered in the otorhinolaryngology clinic. It is important to

identify these patients so that appropriate avoidance measures can be instituted. Generally, these patients are managed in specialist allergy clinics where appropriate testing can be performed and advice about diet provided.

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