Atopy

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Atopy (pronounced /ˈætəpi/; Greek ἄτοπια - placelessness, out of place, special, unusual) or atopic syndrome is an allergic hypersensitivity[1] affecting parts of the body not in direct contact with the allergen.

<table>
<thead>
<tr>
<th>Contents</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Terminology</td>
</tr>
<tr>
<td>2 Presentation</td>
</tr>
<tr>
<td>3 Causes</td>
</tr>
<tr>
<td>4 Signs and symptoms</td>
</tr>
<tr>
<td>5 Pathology and etiology</td>
</tr>
<tr>
<td>5.1 Genetic predisposition</td>
</tr>
<tr>
<td>5.2 Staphylococcus aureus colonization</td>
</tr>
<tr>
<td>5.3 Other allergic diseases</td>
</tr>
<tr>
<td>6 Symptoms</td>
</tr>
<tr>
<td>7 Treatments</td>
</tr>
<tr>
<td>8 See also</td>
</tr>
<tr>
<td>9 References</td>
</tr>
<tr>
<td>10 External links</td>
</tr>
</tbody>
</table>

Terminology

The term "atopy" was coined by Coca and Cooke in 1923.[2][3]

There is some controversy over the terminology.

Many physicians and scientists use the term "atopy" for any IgE mediated reaction (even those appropriate and proportional), but many pediatricians reserve the word "atopy" for a genetically mediated predisposition to an excessive IgE reaction.[4]

Presentation

Atopy is a disease characterized by a tendency to be “hyperallergic”. Atopy is a word taken from the Greek meaning “special” or “unusual”. A patient with atopic allergies has atopic eczema or atopic dermatitis since infancy. Atopic eczema is an extremely itchy skin condition with a hallmark rash that appears most often over the flexural regions (e.g., back of knees, crook of elbows) but can involve almost every region of the body. Crusty, scaly, flattened, erythematous lesions of atopic eczema can appear almost everywhere, but are worse in certain areas or after exposure to certain irritants or allergens (e.g., washing hands with a perfumed or otherwise...
allergenic soap, wearing a wool or scratchy sweater or skirt, rolling across freshly cut lawns). The single most important feature associated with atopic eczema lesions is that they are extremely itchy, and the itch can occur even before the lesions erupt on the skin and are visible.

Causes

It is localized immediate hypersensitivity reaction to an allergen. It may involve eczema (atopic dermatitis [AD]), allergic conjunctivitis, allergic rhinitis and asthma. There appears to be a strong hereditary component. One study concludes that "the general risk of developing AD (3%) and atopy (7%) increases by a factor of two with each first-degree family member already suffering from atopy."[5] Environmental factors are also known to play a major role and the 'hygiene hypothesis' is one of the best paradigms available to date to explain the steep rise observed in atopic diseases. This supports that it is the excess 'cleanliness' of our environments that has led to the decline in the number of infectious stimuli that are necessary for the proper development of our immune system.[6] Many studies also suggest that the maternal diet during pregnancy can be a causal factor of atopic diseases (as well as asthma) in offspring, suggesting antioxidants, certain lipids, and a Mediterranean diet as prevention. [7]

Signs and symptoms

Patients with atopic eczema usually develop what is referred to as the “allergic triad” of symptoms i.e., eczema, hayfever, and asthma. They also have a tendency to have food allergies, and other symptoms characterized by their hyperallergic state. For example, eosinophilic esophagitis is found associated with atopic allergies. Atopy and atopic eczema can be considered a genetic disease because of its strong genetic component, but atopy does not segregate like an autosomal dominant trait. There are certain environmental factors that contribute to its appearance in infants and children, but the underlying cause is a genetic tendency to be hyperallergic. Atopic eczema cannot be prevented in infants because of its genetic origins.

Atopic syndrome can be fatal for those who experience serious allergic reactions, such as anaphylaxis, brought on by reactions to food or environment.

The individual components are all caused at least in part by allergy (type I hypersensitivity reactions). These responses appear after the body is exposed to various allergens, for example specific kinds of food, pollen, dander or insect venoms. Although atopy has various definitions, most consistently it is defined by the presence of elevated levels of total and allergen-specific IgE in the serum of patient, leading to positive skin-prick tests to common allergens.

The multicenter PARSIFAL study in 2006, involving 6630 children age 5 to 13 in 5 European countries, suggested that restrictive use of antibiotics and antipyretics, are associated with a reduced risk of allergic disease in children.[8]

Pathology and etiology

Genetic predisposition

There is a strong genetic predisposition towards atopic allergies, especially on the maternal side. Because of the strong familial evidence, investigators have tried to map susceptibility genes for atopy. These have been reviewed,[9][10] but essentially genes for atopy tend to be involved in allergic responses or the immune system.
**Staphylococcus aureus colonization**

Patients with atopic eczema often improve with the administration of antibiotics or bleach baths (half a cup of bleach per tubful of water) to control bacterial colonization on the skin. Filaggrin mutations are associated with atopic eczema, and may contribute to the excessive dryness of the skin and the loss of the barrier function of normal skin.[11] It may be possible that the filaggrin mutations and the loss of the normal skin barrier expose crevices which make it possible for *Staphylococcus aureus* to colonize the skin.[12] Atopic eczema is often associated with genetic defects in genes which control allergic responses. Thus, some investigators have proposed that atopic eczema is an ALLERGIC response to increased *Staphylococcus aureus* colonization of the skin.[13] A hallmark indicator of atopic eczema is a positive “wheal-and-flare” reaction to a skin test of *S. aureus* antigens. In addition, several studies have documented that an IgE-mediated response to *S. aureus* is present in patients with atopic eczema.[14][15] The unmistakable improvement in atopic eczema observed with antibiotic administration or bleach baths help correlate the hypothesis that *Staphylococcus aureus* colonization is critical to the appearance of atopic eczema.

**Other allergic diseases**

Other allergic conditions such as asthma, allergic rhinitis (hayfever), conjunctivitis, chronic sinusitis, eosinophil esophagitis and food allergies are part of the atopic syndrome.

**Symptoms**

Some symptoms, from an atopy questionnaire[16]:

- Cracks in the skin under the earlobe
- Eczema
  - In elbow flexures and/or hollow of the knees
  - Nipple eczema
  - Neurodermatitis
  - Subtype Dyshidrosis
- Keratosis pilaris
- Perlèche
- Conjunctivitis
- Chronic or seasonal rhinitis

**Treatments**

**Corticosteroids:** For years, there was no treatment for atopic eczema. Atopy was believed to be allergic in origin due to the patients’ extremely high serum IgE levels, but standard therapies at the time did not help. Oral prednisone was sometimes prescribed for severe cases. Wet wraps (covering the patients with gauze like a mummy) were sometimes used in hospitals to control itching. However, a true medical miracle occurred in the 1950s with the discovery that corticosteroids could be used topically in creams or ointments for atopic eczema and other conditions. Thus, the use of topical steroids avoided many of the undesirable side effects of systemic administration of corticosteroids. Topical steroids control the itching and the rash that accompanies atopic eczema. Side effects of topical steroid use are plentiful, and the patient is advised to use topical steroids in moderation and only as needed.
**Immune modulators**: Pimecrolimus and tacrolimus creams and ointments became available in the 1980s, and are sometimes prescribed for atopic eczema. They act by interfering with T cells, but have been linked to the development of cancer.

**Avoiding dry skin**: Dry skin is a common feature of patients with atopic eczema (see also eczema for information), and can exacerbate atopic eczema.

**Avoiding allergens and irritants**: See eczema for information.

**See also**

- Selective ultraviolet phototherapy

**References**

2. Coca AF, Cooke RA. (1923) On the classification of the phenomenon of hypersensitiveness (http://www.jimmunol.org/cgi/content/abstract/8/3/163) J Immunol
External links


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