Allergy and pollinosis

Alergia i pyłkowica

MAGDALENA CZARNECKA-OPERACZ

Departament of Dermatology and Allergic Diseases Diagnostic Center, University of Medical Sciences, Poznań, Poland

Abstract

Allergic diseases are considered to be one of the most important contemporary health problems. The prevalence of these diseases is increasing regardless the age groups or environmental conditions all over the world. Therefore there is a strong need for a better understanding of mechanisms underlying allergic respiratory or skin pathology. Unfortunately there are still some questions to be answered before our knowledge will be satisfactory and our treatment will be effective in all clinical cases. This short paper presents some basic information on allergic inflammation in the case of respiratory and skin allergic diseases. It should be stressed that a close cooperation between clinical practitioners and aerobiologists in terms of pollination monitoring is absolutely crucial for a good prophylactic approach in allergology.

Key words: *allergic reaction, IgE, asthma, rhinitis, atopic dermatitis.*

Streszczenie

Choroby alergiczne stanowią jeden z ważniejszych problemów klinicznych współczesnej medycyny. Częstość ich występowania niestety wzrasta we wszystkich grupach wiekowych i niezależnie od warunków środowiskowych praktycznie na całym świecie. Dlatego też istnieje bezwzględna konieczność lepszego poznania mechanizmów warunkujących rozwój schorzeń alergicznych, manifestujących się zarówno objawami ze strony układu oddechowego, jak i skóry. Niestety, nadal pozostaje wiele niejasności i wątpliwości wymagających dalszych intensywnych badań zanim wiedza nasza w ww. zakresie będzie kompletna i będziemy mogli zaproponować pacjentom zawsze skuteczną terapię. W niniejszym artykule przedstawiono skrótowo podstawowe informacje dotyczące alergicznego procesu zapalenia toczącego się w alergicznych schorzeniach układu oddechowego i skóry. Należy wyraźnie podkreślić, że ścisła współpraca pomiędzy lekarzami praktykami a aerobiologami w zakresie, np. monitoringu pyłkowego jest absolutnie konieczna dla właściwego funkcjonowania profilaktyki alergologicznej.

Słowa kluczowe: *reakcja alergiczna, IgE, astma, nieżyt nosa, atopowe zapalenie skóry.*

(PDiA 2003; XX, 4: 230–234)

Extensive epidemiological evidence from all over the world has recently been accumulated indicating that allergic diseases do affect up to 25% of the whole population and there is an increasing tendency in any age, all races and social classes.

The better understanding of the mechanisms responsible for allergic diseases and an obvious progress in the field of allergology and clinical immunology suggest several hypothesis for explaining the allergy epidemics observed in the last three decades. These include the more sterile living conditions (sterile foods, vaccinations, antibiotics etc.), changes in the environment, but also industrial pollutions and western life-style.

Certainly allergic diseases because of their chronicity, association, progression from one to another and potential severity in certain cases represent one of the most relevant causes of an impaired quality of life and risk for individuals as well as one of the most important socioeconomic problems of the contemporary world. Therefore the approach to allergic diseases in the new millennium is not only the necessity for research but also for unified profile of teaching and health care in different countries [1].

An IgE-dependent allergic reaction is characterized by an early-phase and late- phase reaction (fig. 1.). The early phase involves the release of histamine and other proinflammatory mediators. In the airways these lead, within seconds or minutes to vasodilatation, increased permeability and symptoms of nasal discharge, sneezing, as well as bronchoconstriction [2]. Skin symptoms include itch, burning sensation, erythematous and urticarial lesions or

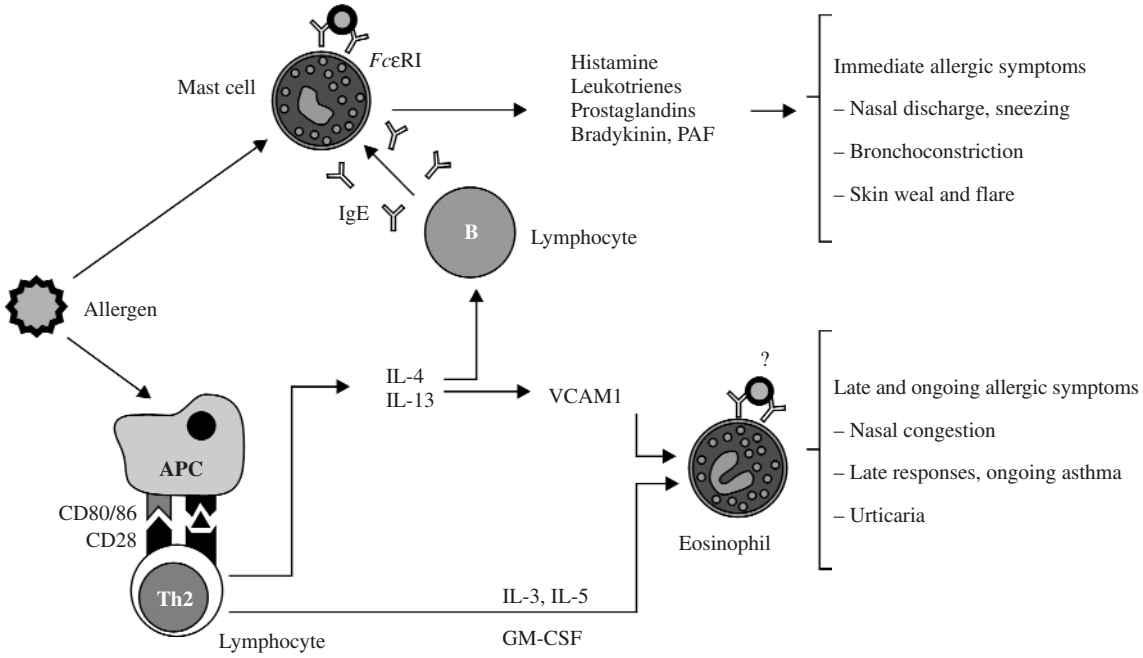

Fig. 1. The IgE-dependent allergy cascade

angioneurotic edema. Due to exposure to allergens we can also observe exacerbation of skin lesions in atopic dermatitis (AD) presenting acute eczematous morphology [3].

During the early-phase reaction mast cells release histamine and other preformed mediators such as hydrolases, proteoglycanes, prostaglandins (e.g. PGD2), leukotrienes, platelet activating factor, bradykinin, and various cytokines (TNF-alfa, IL-4, IL-5, IL-6, IL-10 and Il-13). Proinflammatory cytokines and other mediators initiate a complex network of inflammatory events such as activation of Th2 cells (which release Il-13, IL-4, and Il-5), increased expression of adhesion molecules, and further migration and activation of various inflammatory cells like eosinophils (fig. 2.). These result in the late and persistent allergic problem of nasal congestion, asthmatic bronchial remodeling or skin infiltrations in chronic urticaria and AD.

One of the most important proinflammatory effects of mast cells is to up-regulate or cause the de novo expression of adhesion molecules on the surface of endothelial cells (selectins) and epithelial cells (integrins). These adhesion molecules are involved in the recruitment of inflammatory cells (eosinophils, basophils, neutrophils) from the circulation to the site of the allergic reaction. Specific adhesion molecules favour the tether/roll of inflammatory cels towards the epithelium (e.g. vascular cellular adhesion molecule-1: VCAM-1, P-selectin and L-selectin), arrest of inflammatory cells to the epithelium (CD 18 integrin, intercellular adhesion molecule-1: ICAM-1 and VCAM-1) and diapedesis (transmigration) through the epithelium [4]. Experimental data indicate that inflammatory cells begin to migrate to the mucosa (nasal, bronchial, conjunctival) approximately 30 min after specific challenge, continue to increase during the following 24 hours, and then slowly subside. But up-regulation of expression of the ICAM-1 molecule is evident on the conjunctival and nasal epithelium of allergic patients even when they are asymptomatic [5]. This forms the basis for a theoretical mechanism of the persistent inflammation that continues in the absence of symptoms when a subthreshold exposure to the allergen persists.

There is a strong association between asthma and allergic rhinitis confirmed by cross-sectional epidemiological studies. Large longitudinal studies revealed that allergic rhinitis usually precedes the onset of asthma. Therefore allergic rhinitis is defined as a risk factor for the development of asthma but also rhinitis appears to be an early stage of combined allergic airways disease.

Increased understanding of the nasal and pulmonary allergy cascade has stressed the appreciation of allergy as a systemic disease and the role of early systemic intervention. Recognizing the combined association of nasal and pulmonary allergy is one of major developments

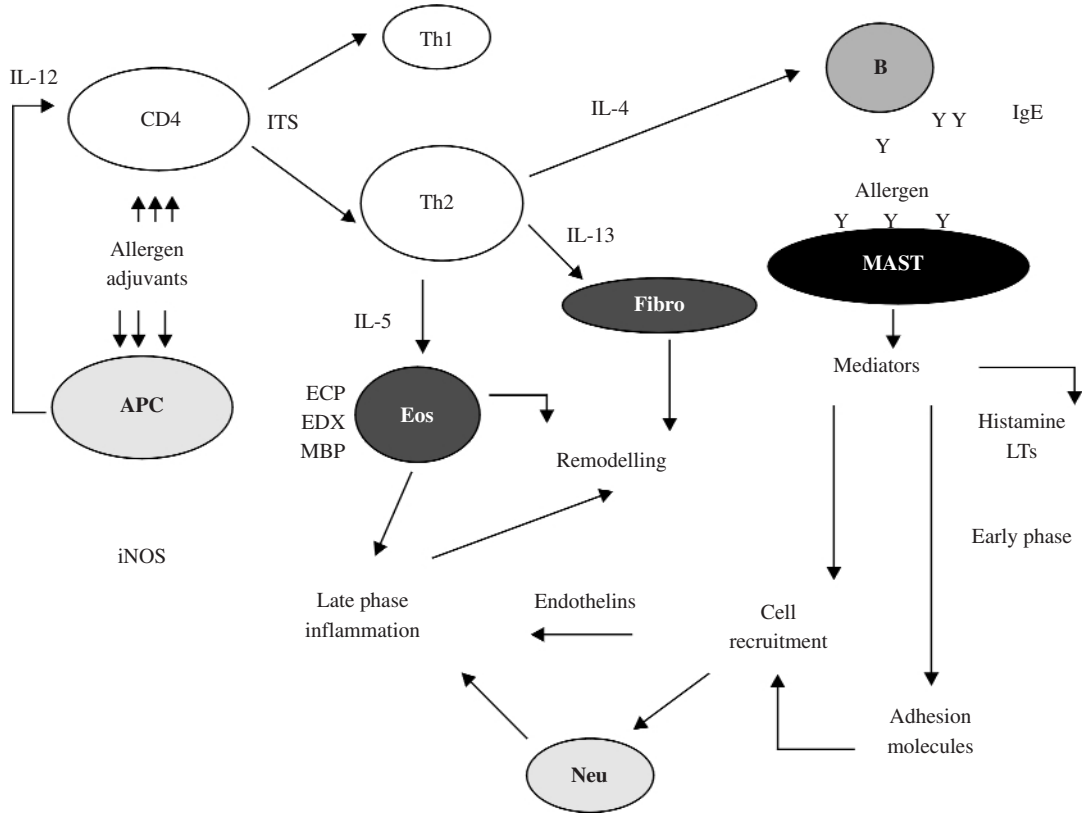

Fig. 2. The IgE-dependent allergic reaction

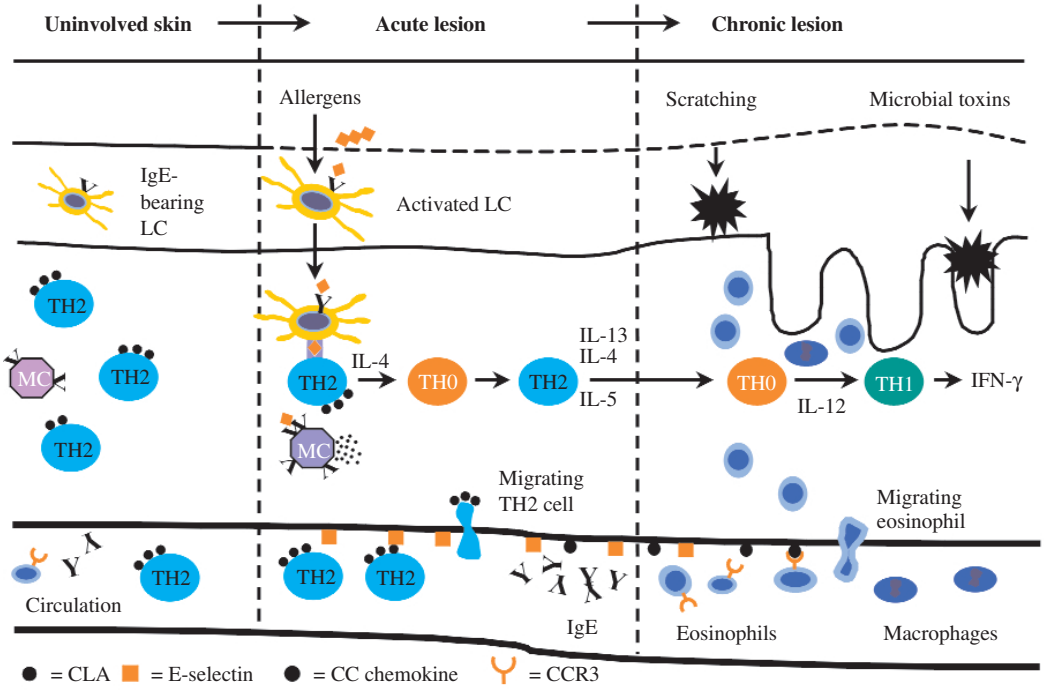

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Fig. 3. Atopic dermatitis-immunological disturbances

in the field of allergic respiratory diseases. It has finally led to introduction of several new terms like *allergic rhinobronchitis, united airways disease* or *combined allergic rhinitis and asthma syndrome* (CARAS) [6]. As recommended by the evidence-based document.

Allergic Rhinitis and Its Impact on Asthma patients with persistent rhinitis should be evaluated for asthma; patients with persistent asthma should be evaluated for rhinitis and a proper therapeutic strategy should combine the treatment of upper and lower airways in terms of efficacy and safety.

The problem of IgE-mediated allergic reactions is even more complicated in case of AD which may be present already in the early childhood of our allergic patients. About 80% of patients with AD may be classified as allergic type of the disease and exacerbations of skin lesions occur after exposure to either food (early childhood) or airborne allergens (both childhood and later). The etiopathogenesis of this disease is partially unclear and is still being investigated (fig. 3.). In case of acute skin lesions there is a clear predominance of TH2 activation while in chronic inflammation an IL-12 dependent switch towards TH1 predomination occurs [7]. Unfortunately patients with AD are in the risk group for asthma development therefore they should be properly diagnosed in terms of IgE-mediated allergy and prophylactic procedures should be introduced as soon as possible. Obviously we can expect situations when a patient with AD is presenting allergic rhinitis sympthoms and finally asthmatic problems arrive but this should be exceptionally rare as our knowledge on multisystemic allergic inflammatory processes is accumulating.

In conclusion I would like to stress that heredity seems to be very important in the pathogenesis of atopic diseases but apart from genetics there are other factors of different origin which influence the onset and the course of those health problems. Numerous genetic and environmental factors interact together giving rise to allergic diseases such as pollinosis, allergic rhinitis, urticaria, asthma or atopic dermatitis.

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The 6th European Course On Basic Aerobiology,
Poznan, Poland
M. Czarnecka-Operacz