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Respiratory Manifestations of Food Allergy in Babies with Atopic Dermatitis: A Prospective Study in 395 Children

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Abstract Background

Nobel Prize for Science Abstract Background Scientific evidence accumulated in recent years indicates that food and inhalant allergens can trigger atopic dermatitis (AD). It may be hypothesized that, also in AD, the allergens could induce a cutaneous hyper reactivity analogous to the bronchial hyper reactivity (BHR) described in allergic patients with asthma. Nonspecific stimuli can therefore trigger and worsen the skin lesions. Eosinophils, as in asthma, seem to play an important role in inducing and maintaining the skin lesions.

Objective: Taken together, these data suggest that in AD there exists a vicious circle, by which immunologic and non immunologic factors act in various ways and at different levels triggering different, though synergistic, reactions to initiate, amplify and maintain the chronic skin lesions characteristic of the condition.

Patients and methods: We have prospectively 395 atopic children attending our Division because they were affected with AD, and found 213/395 babies affected contemporaneously with AD and respiratory allergy. The diagnosis of atopic diseases in the children was done according to family and clinical history, physical examination and positive SPTs and/or RAST to the most common inhalant and/or food allergens Results. Of them 14 babies experienced a positive OFC (open challenge test) to different foods (9 cow milk CM , 5 hydrolysate formulas, HFs and one fish, but we stress that three of them reacted to one drop or two ml of CM) Positivity of family history and elevated total IgE confirm that AD is a genetic disease.

Conclusion: The data confirms the studies that have suggested that allergens in turn could elicit respiratory symptoms that can be distinguished as food induced asthma and asthma with food allergy (FA). In summary, FA provokes wheezing in a small, but significant number of children suffering from AD and asthma, thus confronting pediatricians with one of the most demanding challenges

Keywords: Atopic dermatitis; Asthma; Food allergy; Family history; IgE antibodies; Atopic children; Male sex; Cross reactions; Very small doses of cow milk

Definitions: Food induced asthma: Asthma caused by adverse reactions to foods, what- ever mechanism is involved (immunologic and non-immunologic) [2]; Asthma and FA: children presenting with asthma and allergy to foods, but the causal relationship between asthma and FA is not confirmed [2]; Asthma due to FA: children presenting with asthma and a FA causing respiratory symptoms.

Abbreviations: AD: Atopic Dermatitis; MCs: Cutaneous Mast Cells; SPTs: Skin Prick Tests; PBMC: Peripheral Blood Mononuclear Cell; DTH: Delayed Type Hypersensitivity; ECF: Eosinophil Chemotactic Factor; RAST: Radio Allegro Sorbent Test; ECP: Eosinophil Cationic Protein; EDN Eosinophil Derived Neurotoxin; CM: Cow Milk

Introduction

Atopic dermatitis (AD) is a chronic inflammatory, multi factorial skin disorder. Genetic predisposition appears to be a prerequisite in the majority of cases and the disorder occurs more commonly in children of atopic parents, at an early age, between 0 and one year [3].

Among the common factors able to trigger eczematous skin lesions, so worsening AD in the home environment, it is stressed the contact with inhalant allergens and in particular with Dermatopha- goides pteronyssinus (Der p). In 1930, it was shown that pollens can reach cutaneous mast cells (MCs), and clearly documented the rapid absorption of pollen through the respiratory mucosa and transport to distal ski n MCs [4]. It was then postulated that inhalant allergens play a crucial role in aggravating AD in children aged = 7 years with AD with a stimulating set of experiments trying to demonstrate the pathogenic role of inhaled ragweed pollens in AD [5].

In 1961 Raika [6] confirmed these findings, showing that very often both skin prick tests (SPTs) and intradermal tests were positive to a panel of aero allergens, also in AD patients without clinical symptoms of respiratory allergy. Subsequently, it was demonstrated that specific IgE (and IgG) levels against the Der p 1 allergen were elevated in several test subjects affected with AD, and not with asthma. These results correlated well with the immediate cuti sensitivity for Der p [7].

Later it was first suggested that even contact with Der p may provoke the AD skin lesions. Patients with AD experienced an eczematous reaction after 48 h following application, through patch tests (PTs) on mildly abraded skin, aqueous extract s of inhalant allergens [8].. Skin biopsies obtained at the positive test sites have shown a cell infiltrate formed by peripheral blood mononuclear cell (PBMC) and neutrophil infiltration, and a significant increase in the number of basophils and eosinophils. This type of infiltrate was ascribed to a variant of delayed type hypersensitivity (DTH) reaction containing basophils following local transfer of immune serum but not IgE antibody [9]. A DTH reaction was reported on not manipulated skin of AD patients following PTs with inhalant allergens 48-72 h after epicutaneous testing.

Biopsy specimen obtained at these sites have shown a characteristic PBMC infiltrate [10] similar to the findings reported by Leung et al. [11] in patients with AD (T lymphocytes, eosinophils, Langherans cells - LC, MCs, and basophils). At variance with the Mitchell et al. report [9], the number of MCs and basophils in the biopsy test sites was never higher than 15% of the total cell in filtrate. Other workers [12] have reproduced typical AD lesions on apparently non manipulated skin of subjects with AD by repeatedly applying an ointment containing Der f (Dermatophagoides farinae), thus documenting the percutaneous entry of Der f into the stratum corneum, the epidermis and the dermis.

The authors succeeded in provoking the lesions only by a previous skin scratch and any- how in typical areas, and also hypothesized that AD, rather than being a primary eruption, may result from various and repeated stimuli, and that either type 1 or type IV hypersensitivity are important for its genesis. Decided- ly 15 minutes after the first percutaneous challenge with allergen, a type I urticarial reaction developed in the test patients and only following repeated challenges an eczematous type IV reaction occurred. Positive reactions to PTs have been observed [13] in patients with AD 24-48 h after PT application. Biopsy specimen showed that positive reactions, started by triggering MCs even after 20 minutes, were followed by an infiltration of eosinophils into the dermis 2-6 h after patch-testing.

Positive reactions were clinically similar to that seen in AD skin lesions (erythema with infiltration and/or papules). Immuno staining with antibodies against granular constituents of the eosinophils revealed that infiltrating cells were in an activated state with- out pert of their granular contents. Histologically, there was a predominance of cationic proteins, including ECP (Eosinophil cationic protein) ed EDN (Eosinophil derived neurotoxin). At 24 h eosinophils also appeared in the epidermis but were reduced in number and not activated. At 24-48 h were present in the dermis also T cells, LCs, in- definite cells and MC.

In an attempt to explain the pathogenesis of positive reactions following PTs [14] it was hypothesized that immediately after epicutaneous application of inhalant allergens, part of the allergens penetrate the epidermis, bind IgE antibodies on dermal MCs and induce an immediate type reaction. MCs release eosinophil chemotactic factor (ECF) and, as a consequence, the attracted and in- filtrated eosinophils are in an activated stage. Another part of allergens may bind IgE molecules on epidermal LCs and a T-lymphocyte predominance may en- sue due to the antigen-presenting LC ability, thus causing the PT positivity after 24-48 h. Activated eosinophils which have lost their granular contents are seen in PT lesions: electron microscopy showed that some epidermal eosinophils were in close contact with LCs, thus suggesting a cell-cell interaction [15]. Consequently, eosinophils, as in asthma, seem to play an important role in inducing and maintaining the skin lesions.

Norris et al. [16] applied for 5 days 1 ml of a SPT solution containing Der p antigen on normal un abraded skin of adult patients with AD and SPT positive for Der p (antecubital or popliteal fossae) on either clinically un involved skin or with mild eczema, and allowing scratching caused significant delayed local reactions in one third of test subjects. In are as which were initially clinically uninvolved worsening was not significant. Adinoff & Clark et al. [17-19] found positive PTs to a panel of allergens in 18 patients with AD with positive SPTs to these allergens. The removal or the elimination, where possible, of the appropriate allergens exacerbating the eczematous flares to PTs, induced a marked clearing or the resolution of skin lesion in all patients. These results disagree with t he conclusions of Henderson et al. [20], who have found no parallel between the temporal variations of environmental concentration of dust mites they observed, and the variations of the clinical severity of eczema.

They suggest that their report provides evidence for a pathogenesis of delayed type at the basis of AD. Regarding pediatric asthma, it is increasingly appreciated that AD can be associated with respiratory allergy (25-51% of cases) [21]: children with AD can have a latent predisposition to asthma [22], significantly more elevated if they have a smoking mother [23] or an early onset of AD [24]; in addition AD worsens in spring in children allergic to pollens. Studies have also reported that asthma develops in infants aged 0 to 1 year (3). Thirty-five percent of 500 children aged 6 years were asthmatic, 50 % if suffering from severe AD, and only 15% if from mild AD [25], showing that asthma starts earlier and more frequently in children affected with more severe manifestations [24,26].

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In the 14 studies [26-37], the mean asthma incidence is = 46.8%, but in highly selected pediatric cohorts is between 66% [37] and 79% [34]. In these babies BHR can be present until 95% of cases, while PD20 (provocation dose 20) is m mol 0, 22 in children with AD and asthma and 2, 10 in those without association [38]. The mean age of A D onset is statistically lower also in asthmatic compared with not asthmatic children (p = 0.011) [31], otherwise AD begins within the first yea r and asthma in the following years [39]. From [40-46] it is evident that the prevalence of asthma in children with FA is low (6,3%).

Patients and Methods

We have prospectively followed 395 atopic children, 231 male s and 164 females, aged 1-10 years attending our Division because suffering from AD, selecting the babies affected contemporaneously by AD and respiratory allergy. An equal number of age-matched children (200 males, 195 females) visiting our outpatient department served as controls.

The diagnosis of atopic diseases in the children was done according to the following criteria: clinical history, physical examination and positive SPTs and/or RAST to the most common inhalant and/or food allergens.

We assessed whether the babies were "at risk" of atopic disease because of a positive family history of atopy since one or both parents and/or other sib- lings suffered from asthma, or AD, or allergic rhinitis.

For the diagnosis of asthma, 3 episodes of wheezing without fever were required; For the diagnosis of allergic rhinitis, nasal discharge and/or blockage occurring continuously for at least 4 weeks plus the typical pale aspect of allergic mucosa on rhinoscopy, without any sign of infective rhinitis in other relatives was required. The diagnosis of AD was made according to Hanifin & Rajka criteria [47].

The severity score of AD was recorded with body diagrams according to the SCORAD index [48]; Skin testing was done by the prick method on the volar surface of the fore- arm. The babies were tested with: histamine hydrochloride (1 mg/ml) as a positive control (to ensure the absence of any antihistamine drug interference) and isotonic saline as a negative control. We continued with whole CM protein, casein, lact albumin, egg, wheat, soy, fish, peanut, Dermatophagoides pteronyssinus (Der p), Alternaria alternata, Lolium perenne, Olea europea and Parietaria officinalis (SARM, Roma, Italy).

They were placed on the volar surface o f the forearm as drops through which the skin was superficially pricked with a straight pin [49]. A new pin was used for each SPTs. SPTs were read at 20 minutes and considered positive as follows: + when the wheal was the half of the histamine wheal; ++ when the wheal was equal to the histamine wheal; +++ when the wheal was two-fold the histamine wheal; ++++ when the wheal was more than two-fold the histamine w heal [49]. We took for positive only children with a +++ or ++++ reaction, that is a wheal=3mm with an area=7mm2 (cut-off) We considered as positive only the children with a mean wheal diameter of 3 mm or larger than the negative (saline) control. A positive (histamine, 1:1000) control was performed to ensure the absence of any antihistamine drug interference [50].

Total IgE The total serum IgE level determination was done by paper radio immunosorbent test, and results were expressed in International Units per ml. Specific IgE antibodies and determination of specific IgE levels was done by radio allegro sorbent test (RAST).

RAST results are expressed in RAST Units (PRU = Phadebas Rast Unit) as follows: 1st class = IgE levels < 0,35 IU/ml, 2nd class = IgE levels between 0,35 IU/ml and 0,7 IU/ml, 3rd class = IgE levels between 0,7 IU/ml and 17 IU/ml, 4th class = IgE levels higher than 17 IU/ml. Challenge test The children were previously subjected to a 4-6 week elimination diet period. Nutritionally adequate CM substitutes were employed, such as a soy-protein formula, or Rezza's diet [51]. At the end of this period, when the lesions cleared up, in the children suffering from AD and respiratory allergy were performed 213 OFCs in the hospital under observation in a unit staffed to undertake emergency equipment. The children were tested with CM, 86 with egg and 14 with wheat, soy, fish and peanut. In 23 of them the OFC was done for both CM and egg. CM (or an hydrolysate formula (HF) in the recommended ready to eat concentration, further diluted with water to one part per hundred (1:100), or egg, or fish were administered as follows: a drop of CM (or of HF, or of emulsioned raw egg or a corresponding dose of the other foods) was put upon the inner border of the lower lip, and a Further 5 ml of CM (or of HF, or 1 ml of emulsioned raw egg, or a corresponding dose of the other foods) were given after 5 minutes. One hundred ml of CM, or half-boiled egg, or a corresponding dose of the other foods were given after 30 minutes.

The reactions were defined as immediate if the first symptoms occurred within two hours of ingesting the food antigen, and delayed if the first symptoms occurred after two hours. If any symptoms secondary to the OFC were observed, the OFC in the hospital was terminated. After the last administration of the tested food the children were watched for at least 4 hours and then discharged.

Parents were instructed to continue to give the tested food at home (150 ml of CM twice a day or one boiled egg twice a day or a corresponding dose of fish) and to record any reaction and report it at the following visit 7 and 15 days later [43]. Informed consent was obtained from parents of each study and control child. Data were statistically analyzed using the Student t and the X2 tests.

Results

Again we have demonstrated a male predominance among atopic children (p = 0.0001), and p = 0.0383 between controls and the study group. The difference regarding sex statistics was also significant p = 0.0268.

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Family history was positive in 81,2% of children. We found that 213/395 children suffered from both asthma and AD; of them 14 babies (8 males, 6 females aged 2-6 years, median 2.7) experienced a positive OFC (6,6%). Some babies have reacted even to one drop-2 ml of CM). Among the controls, only one child reacted to CM (p = 0.0001). Abnormally elevated IgE levels (e.g. above 2 SD) were observed in 128 out of 395 children with AD (74-908 IU/ml) (32.4%) and in four control children (p = 0.0001). SPT and RAST results in the study group. The statistical analysis has shown a high significant difference for SPTs (p = 0.0001).

Discussion

The 14 asthmatic babies reacting to foods had a median age < 3 years and experienced immediate respiratory reactions when challenged with foods. We stress that one child reacted to a drop and two to two ml of CM. Therefore they are examples of food-induced asthma, according to both the definitions and the clinical characteristics. We can now delineate the different clinical characteristics of asthmatic children with FA. Several children react to CM with respiratory symptoms: this is confirmed by the meta-analysis on the asthma prevalence in babies with AD (46,8%). In particular, respiratory symptoms were induced by a panel of foods with a mean prevalence of 6,3% (range 2-13,1%) and in this study also by HFs and fish. In a very selected cohort, 5/20 babies (25%) have reacted with wheezing to a DBFC with CM and HFs [52].

Heiner syndrome or recurrent pneumonia was reported in children with hypersensitivity to CM [53]. However, additional foods are able t o cause more or less immediate broncho obstructive symptom s, either due to in- halation for example of airborne fish particles [54], or following ingestion, in relation to a challenge test, or exercise-induced, sometimes with a fatal outcome [55, 56]. Well known are some allergens both alimentary and inhalant, including garlic powder, crabs, shrimps and fish, often with common epitopes [57] and partial allergens, such as Compositae pollen and correlated honey, bromeline extracted from pineapple, finally with common epitopes as above alluded to regarding Artemisia with celery and tomato with grass pollens [58]. The prevalence of food-induced asthma in infancy is to be found within the limits of asthma associated with the foods more frequently responsible, in the order peanuts, CM, egg, nuts [44] , CM, egg, peanuts [42, 54], egg, shell- fish and CM (318), fish (104) and “hidden” allergens [60].

Mono sensitization is present in 77% of cases, in a cohort of 163 asthmatic babies with food poly- sensitization were ascertained the following rates of specific Ig E positivity for one or more foods, varying according to age: 4% if <2 year-old, 24% between 2 and 4 years, and 31% if > 4 years [61]. Among children and adolescents are more common the cross reactions: in this group those sensitive to snails presenting with wheezing in 100% of cases are included, who have the highest levels of total IgE [59] and can have an additional complication, a cross reaction with Der p [60]. To explain the pathogenesis of FA in asthma it is envisaged that the uptake from the airways of the ingested food proteins takes place in two ways [1]

a. Intact molecules may traverse the gastrointestinal tract epithelium and, transported by M cells, reach by the systemic rout e the respiratory tract (and the skin).

b. Inhalation of volatile or aerosolized food allergens, which reach the lung by the respiratory route, certainly the less likely, but de- serves an equal interest. To explain the ingress of food allergens by the digestive route, it has been suggested that ingested allergens are able to traverse the intestinal mucosa in whatever place, due to an increased mucosal permeability, pass subsequently into the circulation, thus reaching systemic circulation and invading distant organs, such as the skin and the airways [58]. Such different situations can interfere with food immunogenicity, which can be reduced or increased depending on the cross reactions becoming apparent between food and inhalant allergens [58] In addition common epitopes can be shared by quite different foods, or relate d epitopes may be destroyed, or previously hidden epitopes may be disclosed. Three out of 14 children reacted to HFs, thus suggesting that concealed allergenic epitopes may be released during the hydrolysis methodology.

Three main categories may be delineated

a. A full-blown identity is found between the aeroallergen and the food allergen

b. The aeroallergen and the food allergen are identical, but the responsible food protein is hidden

c. Common epitopes are shared by wholly different foods and inhaled allergens obtained from quite unrelated sources [58].

Most cases of asthma are observed in areas where pollens are prevalent, due to cross reactions with food epitopes. The more significant proof is given by the remission of respiratory symptoms with an elimination diet and relapse following a DBPCFC [1].

At present the diagnosis of food-induced asthma is m ore reliable when it is associated with:

a. Positivity of family history

b. Wheezing in the first months of life, often related to bronchiolitis or other viral infections

c. High concentrations of total IgE (>1.000 UI/l), especially concordant with SPT positivity, presence of anaphylactic symptoms and/or asthma not well controlled with appropriate management

d. RAST positive for foods. In this study, the positivity of family history and elevated total IgE confirm that AD is a

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genetic disease.

The relationship between respiratory and food allergies is becoming progressively well recognized and may rest on the presence of related epitopes on allergenic proteins from different sources, and on an increasing number of cross reactivities of IgE antibodies [58].

A better understanding of such cross reactivities may enhance our knowledge of associated respiratory and food allergies, hence aiding pediatricians to better prevent these allergies. Primary preventive programs should focus on food elimination associated with strict environmental control to have a proven efficacy with regard to later developing asthma [62]. Allergic and immunologic disorders start in the fetus, in the neonate, in the infant, who are the youngest pediatric populations. So very soon severe atopic disorders confront doctors as one of the most demanding challenges [3].

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