

Short communication

Asthma at Pediatric Age

F. Muñoz-López^{*}

Former Head of the Pediatric Immunology Service, Clinic Hospital and Sant Joan de Deu Hospital, Faculty of Medicine, Barcelona, Spain

^{*}Corresponding author: F. Muñoz-López, Former Head of the Pediatric Immunology Service, Clinic Hospital and Sant Joan de Deu Hospital, Faculty of Medicine, Barcelona, Spain, E-mail: 5314fml@comb.cat

Received: July 26 11, 2019; Accepted: August 19, 2019; Published: August 23, 2019

Abstract

Asthma is an immune-allergological process that usually begins in the paediatric age (between 5 and 16 years of age) usually with a family history of allergy. When they do not exist, the onset is usually later and is usually due to the environment in which they live, due to pollution, as it also occurs in adults who work in unhealthy environments (occupational asthma). The allergic predisposition also predisposes to coexistence with other processes, such as eczema, urticaria, rhinitis, sinusitis and conjunctivitis, which usually begin before asthma.

Keywords: asthma, immunoglobulin, genetic, age, IgE

Introduction

70% of allergic patients belong to families in which the incidence of the disease is significant, particularly in first-degree relatives -parents or siblings- which demonstrates the great importance of genetic inheritance (i.e.: the greatest the risk of suffering from an allergic process is that both parents or close relatives suffer from any kind disease of that nature). If both parents are affected, the risk of offspring suffering from the condition ranges between 40% and 70% depending on the coincidence of the allergic processes suffered by the parents. If only one of the parents is atopic, the risk can fall down to 19%, while if there is no atopic predisposition, the percentage of patients at risk of allergic processes is approximately 12% of the population.

In allergic pathology it is clear that the transmission does not follow Mendelian laws, with a gene as the sole responsible. The atopic inheritance could be polygenic (i.e.: several genes intervene in the same individual) each participating in an aspect of the immune mechanism or in the characteristics of the organ in shock. On the other hand, environmental factors can be decisive for the onset of respiratory diseases, causing bronchial inflammation, which can occur both in paediatric age and in adults due to the environment in which they work (occupational asthma). However, there is no doubt that the genetic factor is decisive in the early onset of asthma, that is, in paediatric age, hence genetic studies, including the different phenotypes, have been fundamental for the knowledge of the origin of asthma in childhood [1-5].

Regarding the age of onset, an appropriate diagnostic and therapeutic approach should be observed in children under 5 years of age who present symptoms of respiratory distress, of diverse intensity, more or less continuously or in acute and repeated episodes. In many cases, the dominant symptom is cough, which has been linked to the existence of asthma ("equivalent asthmatic cough"). As respiratory symptoms are common to many processes affecting this system an appropriate differential

diagnosis is required before starting treatment. There are predisposing, risk and trigger factors: pathogenesis of dyspnoea, cough, secretion and bronchial respiration. The inflammatory reaction is the pathogenic basis of asthma, and therefore, anti-inflammatory medication may be the right treatment. However, there is no evidence that inflammation is a permanent occurrence since the onset of the disease, especially in children, under 5-6 years of age [6].

Clinical Cases

Based on these concepts, in a review of 200 stories of patients attended during a period of one month, 123 of them were diagnosed with asthma, 84 with a family history of allergic disease and 39 without them (Table 1).

There is a clear predominance of patients with a family history (i.e.: with genetic inheritance of the alterations of the genetic elements that condition the onset of allergic processes, especially asthma). In those that do not exist such background, possibly the environment in which they live can cause both allergic disorders, such as the inflammation of the airways, particularly in adults, which is known as occupational asthma.

It is known that IgE serum is not elevated in all patients, however, in this review it is noteworthy that in all patients without a family history it is. Similarly, the serum levels of the other immunoglobulins may be elevated or decreased due to their various activities in the allergic or inflammatory reaction. As can be seen, sinusitis is common in these patients, while there is little coincidence of other allergic processes (conjunctivitis, eczema, urticaria).

In summary and as a treatment for all these patients, in addition to bronchodilator and anti-inflammatory drugs, immunotherapy treatment should be mandatory and is effective in most of the cases, except for certain contraindications (e.g.: severity, poli-sensitization, concomitant infections, severe immu-

Table 1. Genetic, immunoallergological and associated pathology characteristics in children with asthma.

	With family antecedents	Without family antecedents
	Male 57 / Female 27	Male 23 / Female 16
Initial age	All between 5 - 8 yrs	9 > 8º año
Eosinophilia	M: 40,3 % - F: 29,6 %	M: 17,3% - F: 50 %
IgE ↑	V: 91,2% - H: 70,3%	All
IgG ↑↓	↑: M:24,5%- F:59,2% ↓: M and F:7%	↑: M:30,4% - F:37,5%- ↓: M:8,6% - F: 6,2%
IgM ↑↓	↑: M:45,6% - F: 0- ↓M and F: 0	↑: M:56,5% - F:62,5% ↓:M:4,3% - F:0
IgA ↑↓	↑:M:15,7% - F:29,6%- ↓:M:5,6% - F:3,7%	↑: M:39,1% - F:18,7% - ↓:M:4,3%-F:18,7%
Conjunctivitis	None	M: 8,6% - F: 0
Sinusitis	M: 42,1 % - F: 37,0 %	M: 34,7 % - F: 25,0 %
Eczema	M: 0 - F: 7,4%	M: 4,3% - F: 6,2%
Urticaria	M: 1,7% - F: 3,7%	M: 4,3% - F: 6,2%
Other immunoallergic processes.	Immunodeficiencies: 2 cases AAS: 1 case	None

nodeficiency, among others) [7,8].

Difference in Asthma by Age

“Respiratory processes that take place in childhood (preschool and adolescence) have a predominant frequency, especially rhinitis and asthma. Family predisposition and the environment define the characteristics of the endotype and the phenotype. Heritage, both of the genes related to bronchial hyperresponsiveness and those related to atopy (production of specific IgE against allergens and hypereosinophilia) are the fundamental basis of those processes that begin at preschool age and continue into adulthood if they do not receive early and etiological treatment. The physiological vagal hyperresponsiveness of the infant; the environment in which it develops, even from the prenatal phase (pregnant smoker); and viral infections are responsible for frequent bronchial processes in the early years that, sometimes, also extend into adolescence. In summary, the

coordination of the endotype and the phenotype has led to the acknowledgement and acceptance of these three tracheobronchial processes: transient early wheezing, non-atopic wheezing, and atopic wheezing/asthma” [9].

There is no doubt that the genetic factor plays a decisive role in the early onset of asthma, due to the known prevalence of allergic processes in close relatives, although it may also be caused by the environment they live in (animals, smokers, etc.). Bronchial inflammation appears later, so the onset of the disease may be later and appears especially in adults working in polluted spaces. In asthma crisis there is a first phase due to constriction of the bronchial smooth muscle caused by the action of mediators that are released from mast cells; an intermediate phase that is maintained by constriction; and a later phase in which, by intervention of different cells, an inflammatory reaction is consolidated. The variable influence of these factors determines the differences at different ages at which the disease begins, as well as its evolution and the beginning in adulthood [9]. No doubt there are differences in early onset asthma and when it appears at a later stage, especially in adults (heterogeneity) in which the basic treatment is anti-inflammatory drugs, although in younger children, immunotherapy is essential, although not always mentioned [10,11].

References

1. Miller RL, Ho SM. Environmental epigenetics and asthma. *Am J Respir Crit Care Med.* 2008; 177: 567-73.
2. Meyers DA. Genetics of asthma and allergy: what have we learned?. *J Allergy Clin Immunol.* 2010; 126:439-46.
3. Ji H, Khurana Hershey GK. Genetic and epigenetic influence on the response to environmental particulate matter. *J Allergy Clin Immunol.* 2012;129:33-41.
4. Thomsen SF, Duffy DL, Kyvik KO, et al. Genetic influence in the age at onset of asthma: a twin study. *J Allergy Clin Immunol.* 2010;126:626-30.
5. Kaplan A, Hardjojo A, Yu S, et al. Asthma across age: insights from primary care. *Front Pediatr.* 2019, 7:162.
6. Muñoz-López F. Asthma in preschool children. Chapter 4. C. Pereira: From childhood asthma to acos phenotypes. Intech 2016.
7. EAACI. Allergen Immunotherapy Guidelines. Translating knowledge into clinical practice. 2017.
8. Tosca MA, Licari A, Olcese R, et al. Immunotherapy and asthma in children. *Front Pediatr.* 2018;6:1-7 (Art. 231).
9. Muñoz-López F. Meaning of endotype-phenotype in pediatric respiratory pathology. Chapter 3, in “Asthma diagnosis and management. Approach based on phenotype and endotype”. IntechOpen, Croatia 2018.
10. Trivedi M, Denton E. Asthma in children and adults. What are the differences and what can they tell us about asthma?. *Front Pediatr.* 2019;7:1-15(Art. 256).
11. Carr TF, Bleecker E. Asthma heterogeneity and severity. *World Allergy Organ J.* 2016;9:41:1-8.

To cite this article: Muñoz-López F. Asthma at Pediatric Age. *European Journal of Respiratory Medicine.* 2019; 1:1.

© Muñoz-López F. 2019.