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Editorial

Role of Inflammation in Asthma

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Asthma is a disease that affects the general population, regardless of age, and is characterized by an inflammatory state at the level of the airways and by structural changes in their tissues: such as the presence of epithelial hyperplasia of the airways. goblet cells, subepithelial collagen deposition, and smooth muscle hypertrophy. The immunopathology of allergic asthma involves both the humoral and cellular responses, which collectively lead to a state of airway hyperresponsiveness. The cells that are involved in this inflammatory cascade of atopic asthma are: airway epithelial cells, the different subpopulations of T and B cells, mast cells, dendritic cells, eosinophils, basophils, macrophages, iNKT cells and platelets, in addition to the network of cytokines, chemokines, and costimulatory and regulatory signals corresponding to each of the cell subpopulations that orchestrate this process [1].

As has been mentioned, the key pathological marker of asthma, despite all the allergic components or mechanisms that trigger it, is airway inflammation. Indeed, the severity of asthma reflects the degree of inflammation even in asymptomatic forms of the disease. Mast cells and eosinophils are considered the main effector cells in asthmatic disease, since these cells are capable of generating a wide variety of mediators that lead to tissue inflammation. Other cells involved are lymphocytes, macrophages, and epithelial cells through cell-cell interactions, but at the center of this inappropriate reaction are CD4 + T cells, especially TH effector cells [2].

Asthmatic inflammation is attributed to an abnormal sensitivity of different components that trigger a cellular response with an increase in TH2 type cytokines and a decrease in TH1 type. In some cases, TH1 cells tend to assist TH2 in initiating the inflammatory response, even though it is the production of TH2-dependent cytokines that directs and maintains asthmatic inflammation and the consequent pathophysiology [3].

TH2 cells are responsible for stimulating the production of IgE in B cells through the production of IL-4, while IL-5 stimulates the differentiation and mobilization of eosinophils to the sites of inflammation, on the other hand, IL– 10 increases mast cell growth and differentiation and inhibits IFN-y production. Likewise, the reduction in IL-12 synthesis influences allergic inflammation and bronchial hyperresponsiveness by reducing IgE production [4].

The inflammatory process is also promoted by histamine released from mast cells. This inflammatory mediator stimulates bronchial constriction and excessive mucus production and has the ability to influence the development of the adaptive response by acting directly on ICDs. Experimental evidence indicates that the binding of histamine to its receptor in CDi, during the initial phase of their maturation, deregulates the production of IL-12 by the CDm, inducing the polarization and increase of TH2 cells by the expression of CD8 [5].

On the other hand, a second mechanism by which histamine promotes inflammation is by inducing the activation of the nuclear transcription factor kB, since this factor activates genes which give rise to proteins that promote inflammation and maturation of TH cells, in animal models and in patients with asthma. The use of histamine receptor antihistamines, such as cetirizine, indicate that the inhibition of NF-κB in the airways decreases inflammation, the expression of inflammatory mediators dependent on NF-κB, the decrease in the production of TH2-type cytokines and of IgE levels [5].

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