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Pathogenesis of Asthma and Asthma management

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Abstract

Asthma is a serious health and socioeconomic issue all over the world, affecting more than 300 million individuals. The disease is considered as an inflammatory disease in the airway, leading to airway hyperresponsiveness, obstruction, mucus hyper-production and airway wall remodeling. The presence of airway inflammation in asthmatic patients has been found in the nineteenth century. As the information in patients with asthma increase, paradigm change in immunology and molecular biology have resulted in an extensive evaluation of inflammatory cells and mediators involved in the pathophysiology of asthma. Moreover, it is recognized that airway remodeling into detail, characterized by thickening of the airway wall, can be profound consequences on the mechanics of airway narrowing and contribute to the chronic progression of the disease. Epithelial to mesenchymal transition plays an important role in airway remodeling. These epithelial and mesenchymal cells cause persistence of the inflammatory infiltration and induce histological changes in the airway wall, increasing thickness of the basement membrane, collagen deposition and smooth muscle hypertrophy and hyperplasia. Resulting of airway inflammation, airway remodeling leads to the airway wall thickening and induces increased airway smooth muscle mass, which generate asthmatic symptoms. Asthma is classically recognized as the typical Th2 disease, with increased IgE levels and eosinophilic inflammation in the airway. Emerging Th2 cytokines modulates the airway inflammation, which induces airway remodeling. Biological agents, which have specific molecular targets for these Th2 cytokines, are available and clinical trials for asthma are ongoing. However, the relatively simple paradigm has been doubted because of the realization that strategies designed to suppress Th2 function are not effective enough for all patients in the clinical trials. In the future, it is required to understand more details for phenotypes of asthma.

Keywords: asthma, remodeling, epithelial to mesenchymal transition, Th2 cells, cytokines, Th17 cells, Th9 cell

Biography:

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