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Role of IL-13 in the pathogenesis of bronchial asthma

Iman H Shehata [1], Manal M Mostafa, Kholood W Ziada, Adel M Saeed

Department of Microbiology & Immunology, Faculty of Medicine, Ain Shams University, Cairo, Egypt.

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Asthma is a chronic inflammatory disease of the lung. Although it is multifactorial in origin, the inflammatory process is believed to be a result of inappropriate immune responses to common aeroallergens in genetically susceptible individuals. As such, it has been reported that Th2 cytokines play a pivotal role in the pathogenesis of disease. The aim of this study was to evaluate the role of IL-13 in the pathogenesis of bronchial asthma. The study was carried out on 71 subjects out of which 54 were asthmatic patients and 17 were normal controls. Patients with bronchial asthma were further classified according to the National Asthma Education and Prevention Program (NAEPP) into 4 groups: intermittent, mild persistent, moderate persistent and severe persistent. IL-13 serum levels were estimated in patients and controls by ELISA. Asthmatic patients showed a statistical significant elevation of serum IL-13 levels (mean = 78.5 +/- 64.5 pg/ml) as compared to controls (mean = 51.8 +/- 24.9 pg/ml). When patients with different degrees of severity were compared, a significant increase in serum IL-13 was found in patients with intermittent asthma (mean = 106 +/- 105 pg/ml) as compared to those with mild (mean = 63.6 +/- 14.7 pg/ml) and severe persistent asthma (mean = 64.9 +/- 29.1 pg/ml). Only patients with intermittent asthma showed a highly significant increase in serum IL-13 than controls. No statistically significant difference was found between patients not on steroids, patients on inhaled steroids and those on systemic steroids. A statistically significant increase in IL-13 serum levels was observed in patients not on steroids compared to normal controls. A significant increase in blood eosinophil counts was found in patients during acute asthmatic attacks as compared to those who were stable. In conclusion, IL-13 is a key cytokine with critical role in the immunopathogenesis of bronchial asthma. Steroids can downregulate IL-13, but inhaled steroids alone might not be able to abolish the airway remodeling process in asthmatics. Although patients with intermittent asthma are asymptomatic in between exacerbations, inflammation and remodeling are ongoing in their lungs.