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Relationship between Asthma and Essential Hypertension

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Abstract

Asthma and hypertension are two important diseases with a major concern. There has been a dramatic increase in the prevalence of asthma and hypertension in the last decades. The cost of the two diseases is substantial. Both environmental and genetic factors contribute to their inception and evolution. Their shared pro-inflammatory pathway may lead us to search for a shared genetic background. In this review, we try to search for a potential genetic link between the two pathologies.

Keywords: Asthma, Hypertension, Pro-inflammatory pathway, Genes

Introduction

Hypertension is an important risk factor for the development of cardiovascular diseases, including coronary heart disease (CHD), myocardial infarction and stroke. It is the result of the interaction between many risk genes with an estimated heritability ranging from 31% to 68%, and with environmental factors such as: dietary salt intake, alcohol consumption, smoking and ethnicity [1,2]. Various family and twin studies have estimated the clinic heritability of systolic blood pressure and the diastolic blood pressure in the range of 15 to 40% and 15 to 30% respectively [3,4]. The sibling recurrent risk of hypertension is in the range of 1.2 to 1.5 [5], which indicates a phenotype with a modest genetic effect. Blood pressure is regulated by a complex network of interactive physiological pathways involving cardiac contractility and vascular tone through renal, neural or endocrine system, extracellular fluid volume homeostasis and any perturbation in these pathways can arise from genetic or environmental factors or both of them [6]. Genome-wide association studies have identified more than 100 quantitative trait loci attributed to essential hypertension across the genome, especially in chromosomes 1, 2,3,17 and 18 [7]. Most of these candidate genes are involved directly or indirectly in the regulation of the blood pressure, especially the genes of the renin angiotensin aldosterone system [8,9]. Some studies have found a possible association between the genes encoding for the components of the Renin Angiotension Aldosteron System (RAAS) such as the angiotensinogen (AGT), the angiotensin converting enzyme (ACE), angiotensin II type 1 receptor (AT1R) and essential hypertension (EH) [10,11]. It has been discovered that the expression of the AGT gene in the liver is regulated by certain cytokines like interleukin (IL)-1, IL-6 and tumor necrosis factor-α (TNF-α). These ones are likely to be the main activation factors in AGT gene expression. They activate the expression of the AGT gene, elevate the concentrations of AGT and ultimately accelerate the development of hypertension besides the special effect of IL-1 and TNF-α which induce proliferation of the smooth muscle blood vessel cells and arteriosclerosis [12]. Additionally, they play an integral role in the coordination and persistence of the inflammatory process in the chronic inflammation of the airways in asthma since they are capable of inducing many of the pro-inflammatory effects. Indeed the increased and abnormal expression of cytokines in airway cells is one of the major targets of corticosteroid therapy, by far the most effective controller treatment for asthma currently available. Many cytokines and chemokines are involved in the pathophysiology of asthma [13]. It has been reported that the cytokine IL-1 is also involved in asthma besides its implication in hypertension. In this study, patients with symptomatic asthma have increased levels of IL-1β in BAL fluid compared with patients with asymptomatic asthma [14]. Another common cytokine: TNF-α, this latter has an important amplifying effect on asthmatic inflammation [15,16]. There is evidence of increased expression in asthmatic airways [17] and IgE triggering in sensitised lungs leads to increased expression in epithelial cells in both rat and human lung [18,19]. Moreover, there is increased release of IL-6 from alveolar macrophages from asthmatic patients after allergen challenge [20] and increased basal release compared with non-asthmatic subjects [14].

Asthma's Cytokines and their Relationships with Variants in Hypertension and Cardiovascular Disease

A recent study has shown that the blood concentrations of the cytokines (IL-1 and TNF-α) in the hypertensive group were markedly higher than those in the control group (P<0.01), while there was no significant difference in IL-6 concentration between the two groups (P>0.05). In addition, there were significant differences in the average concentrations of IL-1 and TNF-α in the AGT gene 235T allele (P<0.01), but not in the AGT gene 235M allele (P>0.05). The findings of this study suggest that hypertension occurs and becomes aggravated easily in individuals who are carriers of the AGT gene T allele when the concentrations of cytokines (IL-1 and TNF-α) in the carriers are elevated. High levels of IL-1 and TNF-α may promote inflammation, higher expression of AGT and Ca2+ movement into the vascular smooth muscle cells in essential hypertension patients [12].

However, other studies failed to find a relationship between cytokines and CAD such as acute coronary syndrome (ACS) which is defined as an inflammatory disease associated with development of atherosclerosis. In this study, they tried to determine the relationship between IL-1 gene family polymorphisms (IL-1RN, IL-1B in positions -511 and +3953) and ACS in the Turkish population; they have implicated a total of 381 people with 117 control subjects and 264 ACS patients. The results showed no significant differences in both IL-1RN, IL-1B (-511 and +3953) genotype distributions and IL-1RN allele frequencies between ACS patients and the control subjects. [21,22].

Conclusions

Asthma and essential hypertension are multifactorial disorders. The fact of having some cytokines in common, may lead us to search a shared genetic background and thus to find common therapies.

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