Expression of genes associated with neurogenic inflammation in pediatric asthma

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Background: Allergic inflammation triggers neuronal dysfunction and structural changes in asthmatic airways enhancing the process of neurogenic inflammation. Important mediators in neuroimmune interactions are neurotrophins, neuropeptides, neurokines and their receptors as well as histamine pathway. They all play an important role in the pathophysiology of allergic asthma. Aim: We hypothesized that altered expression of genes associated with neuronal dysregulation may affect susceptibility to and the course of asthma. The aim was to investigate the expression of 31 genes related to neurogenic inflammation in allergic bronchial asthma. Methods: In the analysis we included 25 asthmatic patients, aged between 6-16 years. Asthma diagnosis was made according to GINA guidelines. The control group consisted of 20 healthy children aged between 8-16 years without allergies and asthma without allergic background. We analyzed the expression of 31 genes associated with neurogenic inflammation from peripheral blood leukocytes by use of TaqMan Low Density Array method. As an endogenous control we used 18S rRNA gene. The target genes involved, among others, neurotrophins (BDNF, NGF, NTF3, NTF4), their receptors, tyrosine kinases, neuropeptides: SP, NKA, CGRP NEP/CD10, histamine pathway (HDC, HNMT, DAO, histamine receptors), neurokines (IL1β, IL6), receptors of ion channels (TRPV1, TRPA1). Results: We did not observe significant differences in the expression of 31 genes associated with neurogenic inflammation. However, we found a trend towards increased expression of neurotrophin 4 gene in asthmatic patients (p=0.081). Conclusions: The expression of analyzed genes does not seem to be significantly altered in asthma.