Rhinovirus and airway allergy

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Rhinoviruses cause the majority of common colds, which often provoke wheezing in patients with asthma. The precise mechanisms responsible for the rhinovirus infection-induced exacerbations of bronchial asthma remain uncertain. However, several reports have demonstrated airway hyperresponsiveness, increases in chemical mediators in airway secretions, such as kinin and histamine, and airway inflammation in patients with bronchial asthma after rhinovirus infection. Rhinovirus infection induces the accumulation of inflammatory cells in airway mucosa and submucosa, including neutrophils, lymphocytes and eosinophils. Rhinovirus affects the barrier function of airway epithelial cells and activates airway epithelial cells and other cells in the lung to produce proinflammatory cytokines, including various types of interleukins, granulocyte-macrophage colony stimulating factor and RANTES, and histamine. Rhinovirus also stimulates the expression of intercellular adhesion molecule-1 (ICAM-1) and low-density lipoprotein receptors in the airway epithelium, receptors for major and minor rhinoviruses. Rhinovirus infection is inhibited by treatment with soluble ICAM-1 and by the reduction of ICAM-1 expression in airway epithelial cells after treatment with either glucocorticoid or erythromycin. Both soluble ICAM-1 and erythromycin have been reported to reduce the symptoms of common colds. Herein, we review the pathogenesis and management of rhinovirus infection-induced exacerbation of bronchial asthma and the relationship between rhinovirus infection and airway allergy.

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