

Involvement of VEGF in nasal inflammation induced by allergen

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Background : Increased vessel number and vascular permeability with vasodilatation are important characteristics of allergic rhinitis. VEGF is one of the most potent proangiogenic cytokine to increase vascular permeability. Eosinophil is the major effector cell in nasal secretion of allergic rhinitis (AR) patients during early and late response induced by allergen challenge. To evaluate the involvement of VEGF in nasal allergic inflammation, we observed the changes of VEGF and IL-8 levels in relationship with eosinophil cationic protein (ECP) and specific antibody responses in nasal lavage fluid of 52 AR patients after housed dust mite challenge. **Methods :** 52 AR patients sensitized to housed dust mite (HDM) were enrolled; 30 had responses. Nasal lavage fluids were collected before and 10, 30, and 60 minutes, 3, 6, and 24 hours after NPT with monitoring symptom scores. ECP levels were measured by CAP system. Specific IgA antibodies to HDM, VEGF, and IL-8 were measured by ELISA. **Results :** Early responses with elevated ECP levels were observed between 10 to 30 min, while late responses were noted between 6 to 24 hrs with increased ECP level. Increased production of VEGF was noted in early and late phase of AR, but IL-8 level was not significantly changed with allergen exposure. Significant correlations were found between VEGF level, and ECP or specific IgA to HDM($p < 0.05$, respectively). **Conclusion :** VEGF production was noted in nasal secretion after the allergen exposure which could augment eosinophilic inflammation in mucosa of AR patients. This study was supported by a grant of the Korea Health 21 R&D project. Ministry of Health& Welfare, ROK(A050571).

The role of the sensitized alveolar macrophage in T cell proliferation

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Background : It is well known that alveolar macrophage (AM), which composes more than 90% of the bronchial alveolar cells, is the first barrier protecting lung from the harmful antigens. However, few different studies suggest that sensitized alveolar macrophage could exacerbate the asthma phenotype such as airway hyperresponsiveness (AHR). The exact roles of alveolar macrophages in asthma have been unknown. Thus we undertook to determine whether sensitized alveolar macrophage could induce T cell proliferation in asthma animal model. **Method :** BALB/C mice were intraperitoneally sensitized by ovalbumin with alum on day1 and 7, followed by challenge with 1% ovalbumin on day 21, 22, and 23. The lung cells were isolated and analysed by FACS analysis. T cells isolated from the bronchial lymph node and bronchial alveolar lavage (BAL) cells were cocultured with or without ovalbumin in vitro in order to determine ability of sensitized alveolar macrophages to induce T cell proliferation. **Results :** After challenge, the percent of CD11chigh/CD11blow lung cells (alveolar macrophage) were not changed, but the MHC II expression of CD11chigh/CD11blow lung cells increased compared to PBS group. The proliferation of T cells increased 3 times in IP group than other groups without stimulation of ovalbumin. Furthermore, the BAL cells of IP group with stimulation of ovalbumin induced 8 times increase of T cell proliferation compared to that of other groups. **Conclusion :** It is suggested that the sensitized alveolar macrophages in BAL could facilitate the proliferation of T cells in asthma animal model.