招待講演

The Effect of Asian Sand Dust on Airway Epithelium of Allergic Mouse Model

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Background and Objectives : Asian sand dust (ASD) which usually occurs in spring season has influenced the public health problems in East Asian people and it also induces numerous socioeconomic problems. Recent studies have shown that ASD has a potential risk of aggravating the upper respiratory tract symptoms and deteriorating pulmonary function in patients with bronchial asthma. However, the exact effect of ASD on the inflammation of allergic rhinitis patients was not known. The objective of this study was to investigate whether ASD can induce the allergic symptoms and activate the allergic inflammatory reaction in allergic mouse model.

Materials and Methods : Sand dust was collected within the season of ASD. A total of 40 male BALB/c mice (6 weeks of age) were divided into 4 groups. Group 1 was exposed to phosphate-buffered saline nebulization alone and group 2 was also exposed to ASD nebulization alone. Mice of group 3 and 4 were systemically sensitized with ovalbumin (OVA) mixed with aluminum hydroxide gel solution and then they were locally challenged with aerosols of OVA. Thereafter group 3 were exposed to OVA-nebulization intermittently and mice of group 4 were exposed to OVA and ASD aerosol. All groups were divided into the following two groups ($n=5$) : nebulization exposure for a week and for two weeks. The allergic symptom scores (sneezing and rubbing) of each mouse was checked. At 24 hours after the last exposure, blood, nasal lavage fluid (NALF) and bronchoalveolar lavage fluid (BALF) were collected. Next, the number of inflammatory cells and the concentrations of mice OVA specific IgE, IgG1, IL-4 and IL-5 were measured by ELISA method. Finally, nasal mucosa specimen was obtained to evaluate the distribution of inflammatory cells by H-E stain and the expression of IL-4, IL-5 by immunohistochemical staining.

Results : The average symptom score in 1 week model was significantly increased in group 4 (33.5 ± 4.5) compared to group 3 (16.1 ± 3.2) ($p < 0.05$). The eosinophil number of BALF was significantly increased in group 4 compared to group 3 in both 1 week and 2 week models ($p < 0.05$). The IgE level was significantly increased in group 4 (27.13 ± 1.7 ng/ml) compared to group 3 (16.5 ± 0.30 ng/ml) ($p < 0.01$) in 2 week model. However, there was no significant difference between the group 3 and group 4 in the IgG1 level. The concentration of IL-4 in serum showed no significant difference between the group 3 and group 4. However the IL-4 level of NAL was significantly increased in group 4 (48.2 ± 22.8 μ g/mL) compared to group 3 (32.0 ± 10.1 μ g/mL) in 1 week model ($p < 0.05$). IL-5 level showed no significant difference between the group 3 and group 4 both in serum and NAL. In H&E stain, the inflammatory cells were increased in group 3 and 4 compared to group 1 and 2. Immunohistochemical staining showed that the positive cells for IL-4 and IL-5 were expressed in epithelial layer and submucous glands. The positive cells were more increased in group 4 than group 3, however there was no significant difference.

Conclusion : In allergic mouse model, ASD has showed that it can activate the allergic inflammatory reaction, and the increased allergic inflammatory reaction was effected with the increased production of cytokines.