The expression of CST1 in eosinophilic chronic rhinosinusitis

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Eosinophilic chronic rhinosinusitis (ECRS) is the inflammation disease of nasal sinuses characterized by significant eosinophilic infiltration and is progressively increasing in Japan. Patients with ECRS have nasal polyps which are refractory and recurrent easily and can have complications; severe hyposmia, asthma, and eosinophilic otitis media. It is very hard to treat ECRS, yet the pathogenesis of ECRS is largely unknown.

Cysteine proteases are widely expressed proteolytic enzymes that play role in inflammatory tissue destruction. The proteolytic activity of these enzymes is controlled by a family of inhibitors known as the cysteine superfamily, and cystatins inhibit cysteine proteases by forming tight but reversible complexes with their target enzymes.

Cystatin SN (CST1) is protease inhibitor and is one of the type 2 cystatine subfamily. CST1 is expressed in the submandibular gland, bladder, and ureter. CST1 has been shown to bind tightly to the cysteine protease, papain, which is a potent allergen, and to inhibit the cysteine protease activity of papain.

Thus, the cystatin family may play protective roles against allergens with protease activity.

In the previous study, we performed a microarray analysis of nasal epithelial cells from seasonal allergic rhinitis (SAR) patients to the Japanese cedar and control subjects. We observed that the expression of CST1 was upregulated specifically in SAR patients during natural allergen exposure. Immunohistochemical staining confirmed the increased expression of CST1 in the nasal epithelial cells of SAR patients. We have been analyzing the function of CST1 in allergic rhinitis. Additionally, we think that CST1 has the important role in eosinophilic chronic rhinosinusitis, which is the representative chronic inflammation disease such as allergic rhinitis.

In this study, samples of nasal mucosa and nasal polyp of ECRS patients and non-ECRS patients were collected and compared the expression of CST1. Furthermore, we analyzed the function of CST1 by using cultured mast cells.