Opinion

Chronic Rhinosinusitis: Time to Revise the Definition and Phenotypes

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Chronic Rhinosinusitis (CRS) is defined as inflammation of the mucosa lining the paranasal sinuses that lasts more than 12 weeks. Patients with CRS are classified into two subtypes: CRS with nasal polyps (CRSwNP) and CRS without nasal polyps (CRSsNP) [1,2]. However, recent evidences indicate that the inflammation reaches beyond the mucosa of the sinus reaching the underlying bones as part of the disease process. Moreover, accumulating evidences indicate the involvement of special molecular events in patients with allergic rhinitis and CRS, pointing to a distinct TH2 pathway driven sinusitis. These findings alert us to consider revising the definition of CRS and to include allergic CRS as a distinct phenotype in the classification of CRS.

Patients with CRS are radiologically assessed with Computed Tomography (CT) scans that often reveal areas of increased bone density and irregular thickening of the sinus walls. This could explain the recurrence of the mucosal disease after surgical treatment and may indicate that these ostetitic lesions act as the source of the chronic inflammation with worse baseline measures of disease severity and inflammation [3-7]. Other investigators found that the incidence of ostetitis reaches 64% in patients with recalcitrant CRS [8].

Patients who suffer from allergic rhinitis are also prone to develop CRS. Here, the cytokine profile is biased towards Th2 profile indicating a distinct immunological picture. Our group has shown evidence of ostetitis in CRS patients with allergic rhinitis and confirmed the presence of a higher grade ostetitis in CRS patients without allergy. There was a higher grade of mucosal inflammation over bone inflammation in both CRS groups. However, there was no correlation between the grade of bone and mucosal inflammation in each studied group [9].

At the mucosal level we also showed differences in the magnitude and role of inflammatory cells recruitment to the nasal tissue of CRS patients with or without allergy [10,11]. For example, we showed that NK cells infiltrated the epithelial layers of nasal tissue only in CRS patients with allergic rhinitis and not in CRS patients without allergy or controls. NK cells were also more numerous in the stroma of the nasal tissue from CRS patients with allergic rhinitis compared with CRS patients with no allergy or controls. This is due to CX3CR1-ligand interaction on NK cells, as a result of the TH2 induced immunological response. On the other hand, another study demonstrated a novel neuroimmune axis involving eosinophil recruitment in CRS patients with allergic rhinitis through CTH2/Vasoactive Intestinal Peptide (VIP) & PGD2 interaction. This further supports a specific type of eosinophilic inflammation in CRS patients with allergic rhinitis. This should not be confused with other distinct phenotypes such as eosinophilic CRS [12].

From the above mentioned evidences it is believed that rhinologists should consider the inflammation of CRS as an inflammatory process that can extend beyond the mucous membrane. The term Allergic Chronic Rhinosinusitis (ACRS) may be included in the classification of CRS as one of the main groups. This will facilitate a proper management plan for this one disease with several different phenotypes.

References