Eotaxins may contribute to both accumulation and elimination of eosinophils in asthma

I read with interest the study by Coleman et al on lumen eotaxins and eosinophils, and epithelial brushing eotaxins in chronic asthma. Authors conclude that eotaxin-2 and -3 may contribute to luminal migration of eosinophils. However, potential roles of such transepithelial cell traffic are not discussed. In a recent editorial, Rosenberg highlights the possibility that luminal migration of eosinophils importantly eliminates these cells from diseased bronchial tissues. (Coleman et al cite an earlier review by Rosenberg et al, dealing with the complex regulation of eosinophil trafficking). A resolving role of luminal migration would complicate interpretation of eosinophil numbers recorded in sputum and broncho-alveolar lavage (BAL) fluid samples. For instance, a negative correlation between lumen and tissue eosinophils occurs at inflammation resolution.

Involvement of eotaxin in luminal migration was implicated in the first experimental in vivo studies in guinea pigs, demonstrating efficient and non-injurious elimination of mucosal tissue eosinophils across the epithelial lining into the airway lumen (ref 3 and references cited therein). In allergen-challenged allergic mice, peak eotaxin-2 and associated eosinophilia, occurred initially in lung tissue and later in BAL fluid. These data agree with a role of eotaxin-2 first in early accumulation of bronchial tissue eosinophils and then in elimination of these eosinophils by luminal migration.

Coleman et al state that their data are consistent with a previous report by Ravensberg et al of ‘increased eotaxin-2 and -3 in the epithelium of patients with asthma following allergen challenge’. Ravensberg et al actually demonstrated pronounced immunostaining of eotaxin-2 and -3, particularly in lamina propria, and sustained eosinophilia in that location. A strong positive correlation between eotaxins (-2 and -3) and both the subepithelial eosinophilia and the magnitude of late-phase reaction was also demonstrated agreeing with roles of eotaxin-2 and -3 in retaining disease-driving eosinophils in the tissue.

Three studies have recorded time course of bronchial lumen and tissue eosinophilia in allergen-challenged patients with asthma: consistently, resolution of the allergen exposure-induced asthma is associated with reduced tissue eosinophilia and increased lumen eosinophilia (references cited in ref. 3). Since apoptosis/phagocytosis of bronchial tissue eosinophils has not been compellingly demonstrated, these data strongly support the view that luminal migration is a major mode of elimination of eosinophils from diseased asthma tissues. Indeed, the luminal migration mechanism may swiftly eliminate several types of cells (eosinophils, neutrophils, mast cells, lymphocytes, dendritic cells) from diseased mucosal tissues.

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