Journal of Clinical and Cellular Immunology

OPEN ACCESS Freely available online

Opinion Article

Impact of Eosinophilic Inflammation in Chronic Rhinosinusitis

Mark Jansen*

Department of Immunology, University of Amsterdam, Amsterdam, The Netherlands

DESCRIPTION

Chronic Rhino Sinusitis (CRS) is a persistent inflammatory condition that affects the mucous membranes of the nasal cavity and paranasal sinuses. It is diagnosed based on symptoms lasting at least 12 weeks, such as nasal congestion, facial pain, olfactory disturbances and nasal secretions. CRS can present in two distinct forms: with polyps and without polyps. The key difference between these two forms lies in their symptoms and inflammatory characteristics. CRS with polyps often involves hyposmia and eosinophilic inflammation, while CRS without polyps is frequently associated with facial pressure, a neutrophilic inflammatory response and tissue fibrosis.

The immune mechanisms behind CRS involve complex interactions between various immune cells and the tissue microenvironment. Chronic inflammation and tissue remodeling are central features of CRS, influenced by both local immune responses and environmental factors. While CRS with polyps is often characterized by eosinophilic infiltration and a Th2-dominant immune response, CRS without polyps tends to present with a Th1 or Th17-driven inflammation and more fibrotic tissue remodeling. Despite these differences, the role of innate immune cells, particularly Natural Killer (NK) cells, in the pathogenesis of CRS remains less understood.

NK cells are fundamental components of the immune system, involved in both innate and adaptive immune responses. These cells are essential for detecting and eliminating infected or transformed cells and contribute to immune regulation through interactions with other immune cells. In CRS, NK cells are thought to contribute to the inflammatory process, but their precise function and maturation in the context of CRS have not been fully studied. It is believed that dysfunction of NK cells may contribute to the development of chronic inflammation and polyp formation, though this hypothesis has not been conclusively demonstrated.

In this study, we examined the involvement of NK cells in CRS by analyzing their distribution, phenotype and functional capacity in various tissues from patients with CRS with and without polyps, as well as healthy controls. Tissue samples were obtained from 49 CRS patients (36 with polyps and 13 without) and 15 control subjects. Flow cytometry was used to identify NK cells and assess the expression of key functional receptors associated with their cytotoxicity and regulatory functions.

Our findings revealed that NK cells were more abundant in nasal polyps compared to peripheral blood and nasal mucosa, suggesting that NK cells are actively involved in the inflammatory environment of CRS, particularly in patients with polyps. Furthermore, the maturation status of NK cells differed significantly between peripheral blood and tissue samples. In the peripheral blood, NK cells predominantly displayed a cytotoxic phenotype, characterized by the expression of Cluster of Differentiation (CD)11b and the absence of CD27. In contrast, NK cells in tissue samples showed a more regulatory or tolerogenic phenotype, with a mixture of CD11 and CD27+ cells. This shift in NK cell phenotype from cytotoxic to regulatory in tissue environments may reflect an adaptation to the chronic inflammatory state found in CRS.

These findings highlight the involvement of NK cells in the inflammatory process of CRS and suggest that changes in their maturation and activation may play a role in the disease's pathogenesis. The decreased expression of activating receptors on NK cells in CRS patients indicates that these cells may be less functional in responding to inflammatory stimuli. This functional impairment of NK cells could contribute to the persistence of inflammation and the development of polyps, particularly in patients with CRS. It is also possible that factors within the tissue microenvironment, such as cytokines or other immune mediators, influence the maturation and activity of NK cells, potentially leading to a dysregulated immune response in CRS.

CONCLUSION

Our study demonstrates that NK cells are involved in the inflammatory process of CRS, with distinct patterns of maturation and activation in different tissue compartments. These findings suggest that NK cells play a significant role in both the initiation and progression of CRS and their functional impairment could contribute to the disease's chronic nature. Understanding the factors that influence NK cell activity in CRS may lead to novel therapeutic strategies for managing this debilitating condition.

Correspondence to: Mark Jansen, Department of Immunology, University of Amsterdam, Amsterdam, The Netherlands, E-mail: m.jansen@uva.nl

Received: 21-Oct-2024, Manuscript No. JCCI-24-36446; **Editor assigned:** 23-Oct-2024, PreQC No. JCCI-24-36446 (PQ); **Reviewed:** 07-Nov-2024, QC No. JCCI-24-36446; **Revised:** 14-Nov-2024, Manuscript No. JCCI-24-36446 (R); **Published:** 21-Nov-2024, DOI: 10.35248/2155-9899.24.15.745

Citation: Jansen M (2024). Impact of Eosinophilic Inflammation in Chronic Rhinosinusitis. J Clin Cell Immunol. 15:745.