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Commentary

Regulation of Eosinophils in Hypereosinophilic Syndrome

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DESCRIPTION

Eosinophils have long been thought to as cytotoxic effector cells with terminal differentiation. Asthma and respiratory allergies, eosinophilic gastrointestinal illnesses, hypereosinophilic syndromes, and parasite infection are only a few of the diseases that have benefited from the more detailed understanding of eosinophil effector functions that has come from recent studies. Interleukin-5 (IL-5) and other cytokines, such as IL-3 and Granulocyte-Macrophage Colony-Stimulating Factor, cause the development of eosinophils, which are granulocytes, from pluripotent progenitors in the bone marrow (GM-CSF). When IL-5 and chemokines from the eotaxin family function together to react, mature eosinophils are discharged into the peripheral circulation and enter tissues. The distinctive characteristics of eosinophils in peripheral blood and tissues include bilobed nuclei, huge specialised granules that contain cytokines, cationic proteins, and enzymes, and expression of the IL-5 receptor and CC-Chemokine Rceptor 3 (CCR3).

Hypereosinophilic syndrome

Since 1968, when Hardy and Anderson coined the term "Hypereosinophilic Syndromes" (HESs) to describe three individuals with severe peripheral eosinophilia and cardiovascular symptoms, it has been a source of debate. In fact, Chusid. included patients with both more benign phenotypes of HES and those with clear evidence of an eosinophilic myeloproliferative neoplasm in their landmark series of patients with HES published in 1984. They did this because they understood that "there is a continuum of hypereosinophilic disease with eosinophilic leukaemia at one pole." Patients who had secondary, curable eosinophilic illness, such as parasitic infections, were disqualified. According to this description, there are a wide range of clinical symptoms of HES, from a disease that is largely asymptomatic to thrombosis and endomyocardial fibrosis.

In a significant multicenter investigation of individuals with HES, the skin, lungs, and gastrointestinal tract were the most frequently affected organ systems at presentation, in that order of decreasing incidence. Although only around 5% of patients exhibited cardiac and neurological involvement at presentation, these problems ultimately materialised in 20% of patients, emphasising the significance of early diagnosis and efficient treatment in these illnesses.

Eosinophils regulations

One key factor affecting blood and tissue eosinophilia is the equilibrium of eosinophil production, trafficking from the bone marrow to the tissues, and apoptosis. The complex interactions between important transcription factors, such as GATA-1, CCAAT/enhancer-binding protein, and PU.1, as well as the presence of eosinophil-promoting cytokines, the most significant of which is Interleukin (IL)-5, are necessary for the regulation of eosinophilopoiesis from CD34+ stem cells in the bone marrow. Eosinophilopoiesis also takes place in extramedullary tissues, especially when allergic inflammation is present. Here, IL-5 released by tissue-resident type 2 innate lymphoid cells plays a significant role in promoting this process. The surface expression of the IL-5 receptor on the human common myeloid progenitor is a crucial and early step in eosinophil lineage commitment and is maintained even in neoplastic eosinophils, which is consistent with IL-5 playing a crucial role in eosinophilopoiesis. In other clinical situations, such as eosinophilia linked with adenocarcinoma, other cytokines, such as IL-2, IL-3, and GM-CSF, appear to be substantially more significant. Murine models imply that there are checks and balances at numerous levels, but the fundamental processes by which eosinophilopoiesis is downregulated are less well understood.

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